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THE MASSACHUSETTS MEDICAL SOCIETY

Section of Surgery

PRESIDING: Dr. Leland S. McKittrick, Boston, chairman; Dr. John M. Fallon, Worcester, secretary.

THE CHAIRMAN: The first part of the program deals with a subject about which there has been a great deal of discussion and talk during the past few years; a subject about which your chairman and your secretary do not know too much. We therefore felt the need of open discussion of this very vital problem and have placed at the beginning of the program two papers concerning immediate or deferred surgery for general peritonitis associated with appendicitis. Dr. Irving J. Walker will read the first one.

IMMEDIATE OR DEFERRED SURGERY FOR GENERAL PERITONITIS ASSOCIATED WITH APPENDICITIS IN ADULTS

IRVING J. WALKER, M.D.*

BOSTON

WITH the present low mortality of the acute unruptured appendix treated surgically, it is apparent that the attention of the profession should be directed toward reducing the incidence and death rate of peritoneal infection associated with acute appendicitis. As a preventive measure, there should be no letup in the program of education of the public regarding the symptomatology of acute appendicitis and the necessity for immediate operation. The laity assumes that appendectomy per se is not a serious surgical procedure, but overlooks the necessity for early operation to prevent peritonitis. In living up to our teachings, there must be no deviation from the dictum that early acute appendicitis demands immediate surgery. But peritonitis as a complication of appendicitis will always be with us; hence the profession could profitably review the end results of the present treatment of the various phases of peritonitis, having in mind the possibility of reducing the mortality rate.

Our interest in the subject of general peritonitis associated with acute appendicitis was aroused in a previous study[1] of appendicitis at the Boston City Hospital for the years 1927 to 1930, inclusive, at which time we found the mortality for general peritonitis to be 33 per cent. Coller and Potter,[2] from a study of reported series, found that the rate varied from 1.3 to 65 per cent, with an average of 28 per cent. We are convinced that low mortality reports for general peritonitis can be attributed principally to two factors. First, in some series a number of cases included for study had a spreading rather than a general peritonitis, and the fatality rate was consequently low. Secondly, any group of cases cared for by one or two surgeons interested in peritonitis will reflect better treatment and hence a lower death rate than a similar group treated by a large number of surgeons.

We agree with Priestley and McCormack[8] that, for greater accuracy and future reference, cases of peritonitis should be placed carefully and without prejudice in their proper grouping depending upon the kind of disease present at the time of entrance to the hospital. We favor the following classification: (1) local peritonitis, a condition found shortly after rupture of the appendix; (2) appendix abscess, a late manifestation of local peritonitis; (3) spreading peritonitis, in which there is no tendency for localization but only a portion of the peritoneal cavity is involved; (4) general peritonitis, where from abdominal examination the infection evidently involves the entire peritoneal cavity. We recognize the difficulty, on occasion, of differentiating the last two types.

To admit that general peritonitis as a compli-

Read at the annual meeting of the Massachusetts Medical Society, Boston, May 31, 1938.

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cation of appendicitis must have a high mortality, but nevertheless demands immediate surgery in all cases, is to adopt a defeatist attitude which is not justifiable. It is generally appreciated that some patients only moderately ill with general peritonitis can be operated on at once with expectation of recovery, while those desperately ill with it will probably succumb to immediate operation.

We now present for consideration certain data

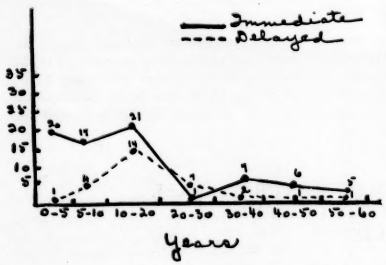

Figure 1. *Incidence according to age groups.*

derived from a study of two groups of general peritonitis treated by immediate and by deferred operation. From July 1, 1932, to May 1, 1938, there were admitted to the Boston City Hospital 5371 patients with acute appendicitis, of whom 105, (2 per cent) had general peritonitis. For purposes of comparing those treated by immediate surgery with those treated by deferred operation, we are omitting the following 3 cases from this study:

Case 1. The patient was moribund on entrance and died in six hours.

Case 2. Diagnosis was not made on admission. An operation for perforated duodenal ulcer was performed twenty-four hours later. The patient died in two and a half days of peritonitis secondary to acute appendicitis.

Case 3. A diagnosis of pneumonia and possible peritonitis was made on entrance. An operation was performed eighteen hours later at which a ruptured appendix with general peritonitis was found. The patient died ten hours after operation.

Seventy-seven cases were treated by immediate operation, and 25 by deferred surgery. Since the latter were treated solely by me, I also include 5 cases treated in private practice during the same period. These 30 cases were consecutive and were not especially selected. In most respects the two groups are quite comparable. It is our impression that those treated by deferred operation were on the whole more seriously sick on admission to the hospital than were those operated on immediately.

Facts of interest in this study include: incidence of general peritonitis according to age and sex, influence of duration upon mortality, complications on and after admission, outline of procedures in those treated by deferred operation and comparison of mortality rates for those treated by immediate and by deferred operation.

It will be noted in Figure 1 that the incidence of general peritonitis, as in acute appendicitis, is most frequent between the ages of ten and twenty.

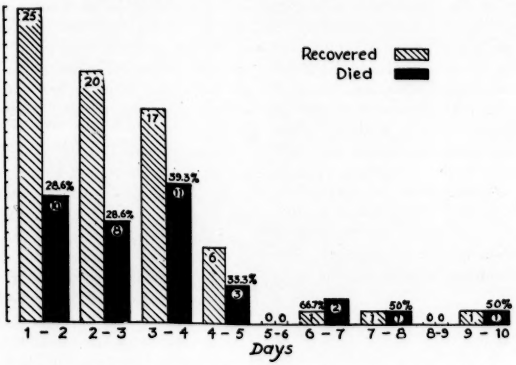

Figure 2. *Mortality in relation to duration of disease before admission to the hospital.*

The youngest patient treated by immediate operation was one year old; the oldest was fifty-nine. The youngest among the group treated by deferred operation was three and a half years and

the oldest fifty-seven. Among the 107 cases there were 66 males and 41 females.

The average duration from onset of symptoms to entrance to the hospital for the entire group was 2.4 days (Fig. 2). While delay in coming to the hospital could for the most part be charged to patients or relatives, in 25 cases a physician was consulted reasonably soon after the onset of acute appendicitis. In none of these cases was a diagnosis of appendicitis made, although it may have been suspected; as a consequence early operation was not advised, with resulting peritonitis. One might conclude that where there exists an acute symptomatology referable to the abdomen, but with the diagnosis in doubt, hospitalization for diagnosis can do no harm and will be conducive to the prevention of peritonitis, if acute appendicitis is present.

Of the 77 cases treated by immediate surgery, 66 had appendectomies, with 25 deaths and 11 had drainage only, with 7 deaths; 60 patients received general anesthesia, with 26 deaths, 15 spinal anesthesia, with 6 deaths, 1 local anesthesia, with recovery, and 1 vinethene, with recovery.

Complications on and after admission in the cases treated by immediate operation are shown in Table 1.

Table 1. *Complications in Cases with Immediate Operation.*

Complications on Admission			
Recovered		**Died**	
Diarrhea	3	Pregnancy	2
		Pneumonia	1
Complications After Admission			
Recovered		**Died**	
Fecal fistula	5	Pneumonia	8
Residual abscess	6	Fecal fistula	3
Pneumonia	3	Residual abscess	2
Intestinal obstruction	3	Paralytic ileus (ileostomy)	2
Wound dehiscence	1		
Empyema	1		
Acute otitis media	1		
Paralytic ileus (ileostomy)	1		

Study of cultures from the peritoneal cavity added nothing to our present knowledge of the bacteriology associated with peritonitis. Extensive anaerobic studies as recommended by Bower et al.[1] were not made.

A review of the records revealed one interesting feature, namely that at the time of entrance to the hospital the group treated by immediate operation could be divided fairly accurately according to prognosis: those moderately ill and those very sick. Of 34 patients classified as moderately ill, 29 recovered and 5 died, a mortality of 15 per cent. Of 43 very sick patients in whom death seemed not improbable 16 recovered and 27 died, a mortality of 63 per cent.

The mortality for the 77 cases treated by immediate surgery was 42 per cent. Figure 3, showing the duration of life after immediate operation, reveals that 14 of the 32 deaths occurred within twenty-four hours after operation. Study of Figure 3 and of individual records suggests that cases with outstanding toxemia may profitably be treated for such time as is necessary to improve the general condition, before operating. We believe that the toxemia is due to infection, dehydration and acid-base imbalance. The situation in respect to toxemia and its treatment is somewhat analogous

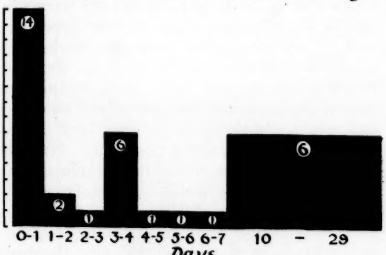

Figure 3. *Duration of life after operation among those operated on immediately.*

to our present treatment of toxic types of acute intestinal obstruction. This observation, made in the previous survey of appendicitis, was our chief reason for the study of general peritonitis treated by delayed surgery.

The cases treated by deferred operation were not only of great interest while under treatment, but also led us to some interesting observations when studied in retrospect. The recent warning of Reid[9] that "the conservative treatment for cases of appendicial abscess and peritonitis is having a somewhat unfortunate effect in that it is being misinterpreted alike by some laymen and doctors," should be taken seriously. He continues: "It is conceivable that the total mortality from acute appendicitis may increase unless the medical profession (not just surgeons) should learn the clear indications for the conservative therapy, and that it has no place in the management of the acute unruptured cases."

We claim no priority in carrying out the principles of the deferred operation in the treatment of peritonitis. Such were first advocated and described by Ochsner.[7] In fairness to him, and to correct any misconceptions of his teachings, he, according to Royster,[10] "never intended his deferred operation as a substitute for operating but only as a preparation for it." Deaver[8] likewise advocated delayed operation where the condition of the

patient was not good. More recently, Guerry[4] stressed the importance of deferred surgery in late cases of acute appendicitis with diffuse peritonitis. Jopson and Pfeiffer[6] are convinced of the value of the Ochsner treatment in certain cases of acute diffuse peritonitis.

Coller and Potter[2] advocate and practice immediate appendectomy for all patients in whom they believe the disease to be limited to the appendix or immediate vicinity, with operation deferred only in cases of spreading peritonitis.

Our criteria for the selection of cases were a history of an immediate attack of acute appendicitis and an abdomen which showed tenderness and spasm throughout. On the basis of the latter criterion, we rejected many cases because they showed extensive but not generalized tenderness and spasm. In doing so we may have omitted some cases of generalized peritonitis. Nevertheless, we are sure that we did not include any case which was not one of generalized peritonitis. We fully appreciate the difficulty of differentiating cases of extensive diffuse peritonitis and those with generalized lesions, knowing that the extent of tenderness and spasm of the abdominal wall probably does not fully represent the degree of the pathologic process within the peritoneal cavity.

In justification for delayed operation, we believe that nature, if unmolested, endeavors to localize infections by the histopathologic process so well described, both clinically and experimentally, by Hertzler.[5] Furthermore, through the production of antibodies and a phagocytic response on the part of leukocytes, infection tends to become self-limited. In addition, it seems likely that drainage of the peritoneal cavity in general peritonitis accomplishes little in removing the entire infection from within this cavity. As against these arguments, it is an accepted principle that perforation of the intestinal tract (in this case the appendix) is best dealt with by immediate surgery, and that pus confined within a cavity should be evacuated.

In observing Ochsner's rule that peristalsis should be inhibited so far as possible in order to localize infection, in our series nothing was allowed by mouth or by rectum and no attempt was made to increase peristalsis until the latter became spontaneously reactivated, as demonstrated by auscultatory evidence and the passage of flatus or feces. Peristaltic action was normally resumed in all except 2 cases where flatus was never expelled; both patients succumbed. Many of our patients on entrance to the hospital were clinically dehydrated and showed an elevated nonprotein nitrogen, with chlorides diminished in varying degrees. It was therefore logical to expect that parenteral fluids and chlorides would have to be used in considerable amounts on entrance and while awaiting peristaltic action. In adult cases it was necessary to give an average of 4500 cc. of physiologic salt solution during the first twenty-four hours. The amount in the younger age group was correspondingly less. After the first twenty-four hours, in the case of adults, the amount of fluids required depended upon whether vomiting was present, and whether decompression of the upper gastrointestinal tract had been carried out by use of the nasal tube and suction. We anticipated the markedly increased loss of chlorides and fluids under these circumstances. In such cases the amount of parenteral fluids given in the form of glucose in normal salt solution approximated the amount vomited or recovered by the Wangensteen method, plus 1000 cc. The total urinary output in twenty-four hours also was used as an index of the amount of fluids required. In some instances there was a temptation to give excessive amounts of sodium chloride without recognizing its possible deleterious effects. Practically, we found that where there was no loss of fluids from the upper intestinal tract, 1000 cc. of physiologic saline solution with 10 per cent glucose, given intravenously morning and night, together with 1000 cc. of 5 per cent glucose in sterile water by clysis in the interval, maintained a normal fluid level and an acid-base balance — a total of 3000 cc. of fluid and 17 gm. of salt.

We early discovered that when the continuous intravenous drip method was maintained for several days thrombosis of veins was likely, and at times we were placed in the predicament of having no veins available when needed. It is our opinion that after the first forty-eight hours fluids should best be administered, not by continuous intravenous drip, but by using alternate veins from time to time. Such a routine was not possible where large amounts of fluids were required within a relatively short interval. In 3 of the 30 deferred-operation cases peripheral edema developed; it proved to be due to a combination of excessive fluids and salt, together with deficient serum protein. This was corrected by reduction of fluids, substitution of glucose in sterile water for salt solution, and blood transfusion. When peristaltic action had established itself, within an average period of four days after hospital treatment, fluids in small and increasing amounts were allowed by mouth, with a diminishing amount of parenteral fluids. If regurgitation recurred, this process was reversed.

Diarrhea after normal bowel movements was not unusual. The reasons for it are as yet not clear to us. In 3 cases severe diarrhea necessitated a return to the use of parenteral fluids for the time being.

Blood examinations were made on entrance and periodically to determine the presence of septicemia and anemia, as well as to study the blood chemistry. There were 4 cases of septicemia, with the following bacteriologic findings: *Bacillus coli; B. coli,* Type 4 pneumococcus; Type 25 pneumococcus; *B. coli, Streptococcus hemolyticus.* Transfusions

Table 2. *Location of Abscesses.*

	NO. OF ABSCESSES
Pelvis	20
Left lower quadrant	7
Right lower quadrant	7
Mid-abdomen below umbilicus	7
Right subdiaphragmatic area	3
Left subdiaphragmatic area	1
Mid-abdomen above umbilicus	1
Right flank	1
Left flank	1
Total	48

were freely used for those cases which showed septicemia or anemia, or in which sepsis was prolonged. In 4 cases there was a low polymorphonuclear count on admission. Satisfactory response followed the use of Pentnucleotide.

X-ray films of the chest were made soon after

Table 3. *Complications in Cases with Deferred Operation.*

COMPLICATIONS ON ADMISSION			
RECOVERED		DIED	
Diabetes mellitus	1	Chronic alcoholism	1
COMPLICATIONS AFTER ADMISSION			
RECOVERED		DIED	
Pneumonia	7	Pneumonia	4
Phlebitis	3	Phlebitis	1
Peripheral edema	2	Peripheral edema	1
Severe diarrhea	2	Severe diarrhea	1
Intestinal obstruction	2	Deep abscess of leg	1
Left hemiplegia (temporary)	1	Purulent pericarditis	1
Pyelitis	1	Septicemia	2
Septicemia	2		

entrance to determine the condition of the lung fields and the position of the diaphragms, knowledge of the latter being useful in comparison with fluoroscopic examinations and plates taken later where subdiaphragmatic abscess was suspected. Morphine was given only in amounts necessary to control pain.

We intended originally not to operate except for the drainage of abscesses or for intestinal obstruction, and to perform an appendectomy only when all evidence of peritoneal infection had subsided. Two patients recovered without the formation of an abscess. In order to prove that the appendices had been the cause of the peritonitis, somewhat against our best judgment, we removed them while the patients were still in the hospital. Both recovered, and each proved to have had a rupture of the appendix. A third patient received an appendectomy at the time of operation for a suspected but nonexistent intestinal obstruction.

Among 28 patients there were forty-eight abscesses. The largest number of abscesses in any one patient was five. The average time from admission to the formation of the first abscess was four days. The location of the abscesses is shown in Table 2.

In addition, a left subdiaphragmatic abscess was found in 1 case that came to autopsy. Possibly the preponderance of pelvic abscesses could be accounted for by the fact that patients were kept in Fowler's position until abscess formation had oc-

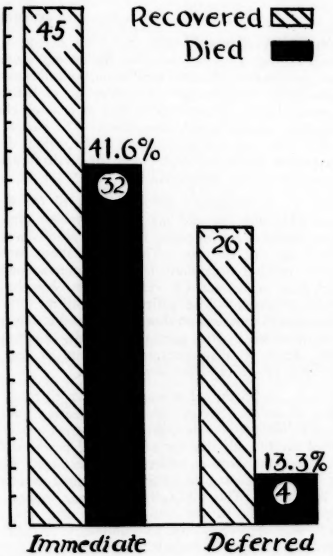

Figure 4. *Comparative mortality in patients treated by immediate and by deferred operation.*

curred. The pelvic abscesses were opened by colpotomy in the female and proctotomy in the male, under spinal anesthesia in the adult and general anesthesia in children. Superficial abdominal abscesses were incised under local anesthesia. The right subdiaphragmatic abscesses were opened by a two-stage transpleural approach under local anesthesia, and the one occurring in the left side through a subcostal incision.

The complications on and after admission are listed in Table 3.

In the 30 cases treated by deferred operation there were 4 deaths, a mortality of 13 per cent, compared with a mortality of 42 per cent in 77 cases treated by immediate operation (Fig. 4).

Data concerning the fatal cases and the causes of death are as follows:

Case 4. A 13-year-old boy was admitted in a state of shock. He had been ill for 10 days, during which time he had been treated for intestinal infection by a physician. The following operations were performed: 9th day after admission, first stage drainage of right subdiaphragmatic abscess; 11th day, second stage drainage of right subdiaphragmatic abscess; 16th day, drainage of left subdiaphragmatic abscess; 24th day, drainage of left lower-quadrant abscess; 27th day, drainage of abscess above umbilicus. The complications were as follows: double lobar pneumonia; deep abscess of leg; septicemia (*B. coli* and Type 4 pneumococcus); phlebitis. The patient died of pneumonia on the 57th day. The septicemia had cleared up. There was no postmortem examination.

Case 5. A 30-year-old man had been ill for 5 days before calling a physician. The following operations were performed: 5th day after admission, drainage of abscess below umbilicus; 6th day, drainage of left lower-quadrant abscess. Complications were as follows: chronic alcoholism; double bronchopneumonia. The patient died on the 11th day. A postmortem examination showed an appendix which had ruptured, a left subdiaphragmatic abscess and cardiac dilatation.

Case 6. A 15-year-old boy had been ill for 4 days before admission, but did not see a physician until just before entrance. An operation for pelvic abscess was performed on the 4th day. The complications were as follows: marked distention; lobar pneumonia; purulent pericarditis; septicemia (*B. coli* and *Str. hemolyticus*); peripheral edema. The patient died on the 5th day. A postmortem examination showed a rupture of the appendix 4 cm. from the tip, general peritonitis, bronchopneumonia, acute purulent pericarditis, edema of legs and septicemia (*B. coli* and *Str. hemolyticus*).

Case 7. A 50-year-old woman had been ill for 4 days before admission. She did not see a physician until just before coming to the hospital. An operation was performed on the 4th day for drainage of pelvic and left lower-quadrant abscesses, under spinal anesthesia. On the way back to the ward the patient complained of difficulty in breathing. This difficulty increased, cyanosis developed, and the patient died 12 hours after operation. Death was apparently due to pulmonary embolism or respiratory paralysis secondary to spinal anesthesia. There was no postmortem examination.

In analyzing these 30 cases with deferred operation, it is fair to state that 5 patients were moderately ill on admission and might well have recovered following immediate surgery. We believe that a large percentage of the other 25 would have succumbed to immediate surgery and were benefited by deferring operation. We allowed the 23 patients who recovered and whose appendices were not removed in the hospital to return home when all signs of infection had subsided, with instructions to re-enter the hospital in three or four months for appendectomy, or at once should any abdominal symptomatology manifest itself. In all these cases the appendixes were subsequently removed.

We were greatly impressed at the time of late appendectomy by the extent of the gross healing that had taken place in the appendixes and by the slight evidence of a recent generalized peritoneal infection, as shown by the small number of adhesions. In 1 case, however, in which an appendectomy was performed four months after discharge from the hospital, a small amount of pus, which proved to be sterile, was found in the region of the appendix. This case also showed more evidence of peritoneal adhesions than any of the others.

It should be understood that the treatment of these cases constituted a clinical study. We do not believe that in practice one should necessarily wait for the formation of an abscess before operating. By treating toxemia before operating, 23 of the 30 cases were, within twenty-four or forty-eight hours, placed in a condition much more favorable for tolerating surgery than they were on admission. The remaining 7 cases, while perhaps not improved by this treatment, were at least not in any more unfavorable condition than they were on admission.

In general, deferred-operation treatment should not be attempted unless the surgeon has a knowledge of the clinical aspects and lesions of general peritonitis and a concept of the physiology of the intestinal tract and realizes the importance of recognizing and treating toxemia due to dehydration and acid-base imbalance. He should have at his command chemical, bacteriological and x-ray laboratories with trained technicians, an intelligent resident staff and medical consultants. He must not allow his emotion to overrule his judgment at any time.

SUMMARY

Early acute appendicitis demands immediate surgery.

Patients with an acute atypical symptomatology referable to the abdomen should be hospitalized for diagnosis.

The incidence of generalized peritonitis among 5371 cases of acute appendicitis was 2 per cent.

Cases of generalized peritonitis can be classified as moderately ill or seriously sick on entrance to the hospital.

Patients moderately ill with generalized peritonitis can be operated on immediately with a comparatively low mortality, while those seriously sick, if treated in this manner, show a high death rate.

Seventy-seven cases of generalized peritonitis treated by immediate surgery gave a mortality of 42 per cent.

A group of 30 cases treated for toxemia and

with delayed operation showed a mortality of 13 per cent.

Deferred operation has no place in the treatment of the acute, unruptured appendix. It should be considered for those patients desperately ill with fulminating peritonitis, only when and if the surgeon is equipped in every way to carry out the necessary procedures.

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THE CHAIRMAN: In talking about this subject with men about the state, or in bringing it up at our staff meetings at the hospital, one is constantly faced with the statement: "The situation in the adult is different from that in the child" or "It is different in the young adult as compared with the older adult." It has therefore seemed appropriate to call on one whose experience certainly exceeds that of any of the rest of us around this community to present this same problem, as they have had it at the Children's Hospital. I ask Dr. William E. Ladd to carry on the discussion.

IMMEDIATE OR DEFERRED SURGERY FOR GENERAL PERITONITIS ASSOCIATED WITH APPENDICITIS IN CHILDREN

WILLIAM E. LADD, M.D.*

BOSTON

THIS paper is based on the impressions gained from thirty years of experience with appendicitis in children, on some statistical data gathered by two of my associates, Dr. Henry W. Hudson, Jr. and Dr. John W. Chamberlain, and on some ideas expressed in the literature.

Some fifteen to twenty years ago many thought that the last word had been said about appendicitis and that the situation was well in hand. This belief has in recent years been proved to be completely erroneous. According to Sperling and Myrick[1] appendicitis causes 20,000 deaths annually in the United States, the highest mortality rate in any civilized nation in the world. The Massachusetts Department of Public Health shows that the number of deaths from appendicitis in children from 1900 to the present day has increased, and that there has been no decrease in the mortality rate from this disease. This department has also shown that from 1926 to 1936 among the chief causes of death in this state appendicitis had jumped from sixth to third place. It is now preceded only by pneumonia and accidents.

With this lamentable situation in both state and nation staring us in the face, it seems imperative that a serious attempt be made to ascertain the causes and provide a remedy.

Read at the annual meeting of the Massachusetts Medical Society, Boston, May 31, 1938.

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It is certainly not inaccurate to state that deaths from appendicitis are preventable, and that the medical profession has fallen down on its job by allowing this large number of deaths to result annually from this disease. It is a fact that deaths from appendicitis uncomplicated by peritonitis are negligible in the Children's Hospital and also in many other well-organized clinics. In the last ten years at the Children's Hospital 300 appendectomies were performed for chronic, recurrent or interval appendicitis, with no deaths. Three hundred and sixty-one operations were performed for acute suppurative appendicitis, 248 without fluid and 113 with cloudy fluid but without gross rupture. In these 361 acute cases there were 2 deaths, — 1 from hemophilia, — a mortality of 0.55 per cent. From these statements it is clear that our dilemma at the present time is largely dependent on late diagnosis and delayed operation.

There are those who consider that appendicitis in childhood is identical with that in the adult and that it can be dealt with in exactly the same manner. Those who consider the child as simply a small-sized adult constantly flirt with and frequently suffer disaster. Although it is true that the disease of appendicitis is the same in the child as it is in the adult, the reaction of the child may be quite different, and the diseases common to childhood from which it must be differentiated are in a very different proportion to those of adults. Anatomically, the only differences between the

child and the adult are that the appendix is relatively larger as compared to the size of the abdominal cavity, that the mesentery may be relatively longer and less fixed, and that the omentum may be shorter and higher. These factors tend to make the maximum point of tenderness more varied in location and the omentum less effective in its function of walling off an infectious process.

The age incidence of appendicitis in children is of importance only in so far as it varies the probability of the occurrence of the disease at different ages. Appendicitis is extremely rare in the first year of life, moderately infrequent in the second year, and from then on becomes common. It is of slightly more frequent occurrence in the period from six to twelve than from one to six years of age.

Some writers have stated that appendicitis is commoner in the months of the year in which respiratory infections are most prevalent. Other writers consider that it occurs more commonly in the summer months when digestive disturbances are commonest. A statistical study of the cases at the Children's Hospital reveals that neither of these theories is borne out by experience there. However, it is not uncommon to find a history of a preceding respiratory infection in cases of appendicitis.

The usual story is one of acute abdominal pain followed by vomiting. The pain in the early part of the attack may be situated in the epigastrium, or more commonly around the umbilicus. It must be remembered that children, especially younger ones, are vague and inaccurate about describing their pain. One should consider that evidence of pain in any part of the abdomen is a danger sign, and appendicitis must be considered as a possibility. Vomiting is a common accompaniment of the abdominal pain, and in a child should be considered by the laity as an indication for seeking competent medical advice, not as an indication for seeking the castor oil bottle and being lavish with its contents. Bower[2] deduced from an analysis of records of twenty-eight Philadelphia hospitals that of patients suffering from acute appendicitis 1 in 80 died who had taken no cathartic, while of those who had been purged more than 1 in 7 died. Whether these figures could be duplicated in other centers is relatively unimportant. There seems to be little doubt that active catharsis causes increased intra-appendicial pressure, with greater probability of rupture and dissemination of the infection.

I hesitate to mention such an elementary subject as the technic of the physical examination of children, and would not do so but for the fact that I have seen both surgeons and medical men of great repute make an examination in such a manner that they cannot possibly gather half the information that they might, and that half very likely is incorrect. It has been my observation that the man who makes a hurried, rough examination in the wrong order is prone to make an unnecessary number of incorrect diagnoses.

The common routine of a physical examination starting with the head, gagging the child with a throat stick, upsetting him with a cold stethoscope, and then applying such deep palpation of the abdomen that it is painful, regardless of the presence or absence of inflammation, is almost valueless.

In the case of a suspected lesion in the abdomen of a child, a careful history must first be taken from the parent, and this supplemented by questioning the child if he is old enough. Next, the child's confidence must be gained before starting the examination. It often requires much patience and time to accomplish this, but it is time well spent. Then everything that can be seen by inspection, such as rate of respiration, color of skin, position of legs, evidence of pain, restlessness, sunken eyes and so forth should be noted. The abdominal examination should precede the general examination. Very gentle palpation should come before deep palpation, and will often give much more valuable information than the latter, which frequently throws the whole abdominal musculature into spasm. Continued steady and slightly increasing pressure will often overcome spasm or fullness of the abdomen when it is due to disease in the chest, whereas it will not do so if the inflammation involves the peritoneum. The palpation of the abdomen should always include a bimanual examination of the kidney region. Abdominal percussion and auscultation should follow the gentle palpation, and finally, deep palpation should be done if necessary.

The general physical examination should follow the abdominal and should always include a rectal examination. If this sequence is followed, I am sure fewer mistakes will be made than when the usual routine is pursued. The rule that children show less on physical examination for a given amount of disease in the abdomen than adults do, though not infallible, is sufficiently often correct to be worthy of mention. It is quite striking that a child with even a ruptured appendix situated in the retrocecal region or deep in the pelvis may show only a very slight amount of muscle spasm or tenderness by abdominal examination.

The laboratory aids to diagnosis which should be done as a routine are the white-blood-cell count, the differential count and the urine examination. The blood examination commonly shows a

white count elevated to 15,000 or 20,000 and a high polymorphonuclear ratio. The urine examination should rule out a lesion in the urinary tract, but it must be remembered that appendicial inflammation adjacent to the ureter may cause a few white and red blood cells to be found in the urine.

The x-ray has been used in some clinics to determine the question of free gas in the peritoneal cavity, thereby refining the diagnosis to one of ruptured or non-ruptured appendicitis. This procedure seems to me to be one of academic interest rather than one of practical importance, but this point of view may be due to lack of personal experience along this line. Our use of the x-ray in cases of suspected appendicitis has been limited to the examination of the chest in occasional patients to rule out the possibility of a central pneumonia undetectable by physical examination.

In childhood the commonest diseases which offer difficulties of diagnosis are pneumonia, infection of the urinary tract and metastatic or idiopathic peritonitis. In the differential diagnosis of these three diseases from appendicitis, one should first be made suspicious by the discrepancy in the temperature. In all three the temperature tends to run higher than in cases of appendicitis. While in these three diseases and appendicitis the onset of the disease may be ushered in by nausea, vomiting and pain referred to the abdomen, it is uncommon for the temperature in appendicitis, even complicated by peritonitis, to run much over 103°F., while in the other three conditions a temperature over 104°F. is common. In pneumonia, although the symptoms may attract one's attention to the abdomen, the higher temperature, the often higher white count, the higher respiratory rate and a careful physical examination and x-ray of the chest should enable one to differentiate the two diseases. In pyelitis, again, the higher temperature, costovertebral tenderness and sometimes the urinary findings enable one to arrive at the correct diagnosis. Metastatic peritonitis, I believe, is impossible at times to distinguish from peritonitis of appendicial origin, but we always attempt to differentiate the two conditions and are correct in most cases. The points of distinction in the order of their importance are a higher temperature, more diffuse tenderness, spasm and distention earlier in the course of the disease and a higher white-cell count; of less importance are a preceding history of respiratory infection or diarrhea.

Other and rarer diseases, which are commoner in the child than in the adult and which may cause difficulties in the diagnosis, are Meckel's diverticulitis, acute mesenteric adenitis, duplications of the alimentary tract, ureteral calculus, urinary stasis due to congenital defects, the various forms of intestinal obstruction and, occasionally, new growths. Cholecystitis and salpingitis occur in the child, but only very rarely. In spite of the variety of conditions in which the symptoms may simulate appendicitis, the errors of diagnosis should not be numerous.

After the diagnosis of acute appendicitis has been made, it seems to me that there should be very little difference of opinion as to the best form of treatment to adopt. However, such is not the case, and there is a considerable divergence of opinion as to when to operate, how to operate, whether to drain, how to drain and what the aftercare should be. This confusion has been considerably added to in the last few years by the resurrection of the Ochsner treatment and the misinterpretation by the medical practitioner of how and when this should be adopted. The question of immediate or delayed operation in cases of appendicitis requires the nicest kind of surgical judgment by a surgeon of experience. For the medical man to assume the responsibility of this decision is a mistaken policy which frequently changes a simple problem to a complicated one and sometimes results in an unnecessary fatality. The function of the medical man should be to call a surgeon either when he sees a patient with appendicitis or when he sees one whom he suspects of having it. If this policy were generally adopted, the problem of peritonitis of appendicial origin would be greatly reduced.

The Ochsner[3, 5] treatment was recommended as early as 1892; it consisted in placing the patient in Fowler's position, applying heat to the abdomen, giving nothing by mouth and performing a gastric lavage two or three times. Ochsner believed that no anodyne was necessary, as the pain would be relieved by the gastric lavage. He did advocate small nutrient enemas during the period of delay while waiting for an abscess to develop. When an abscess developed this was treated by simple drainage. This form of treatment was recommended only for patients having diffuse peritonitis, and Ochsner[4] stated in his book, published in 1907, that it was unsatisfactory in children. His reasons for this belief were that the child was apt to injure himself during the struggle against the lavage or rectal feeding, and that the omentum was too short to protect the peritoneum efficiently. In the light of our present-day knowledge his reasons were undoubtedly partially incorrect. Children tolerate gastric siphonage by a small catheter introduced through the nose quite as well as do adults. The real reason why this treatment does not work well in children is unquestionably due to the resulting dehydration and ketosis, which always cause children to go downhill very rapidly.

The present-day modification of the Ochsner

treatment, as recommended by Coller and Potter[6] and others, should be more successful, as the continuous or repeated intravenous administration of saline and glucose solution quite effectively prevents or overcomes dehydration and acidosis. This does not alter the anatomic variations of the child, or the fact that children localize a peritonitis less effectively than do adults.

I believe that in the last few years medical teaching has swung too far to the conservative side. I frequently see medical students, pediatricians and general practitioners laboring under the erroneous and dangerous idea that all cases of acute appendicitis should be quieted down and later have an interval operation.

It is our practice at the Children's Hospital to operate on the great majority of patients with appendicitis, including those with peritonitis, as soon after their admission to the hospital as they can be properly prepared by the administration of parenteral fluids and so forth. We limit the delayed operation to the occasional patient who is profoundly toxic, markedly dehydrated and very distended, and who has sunken eyes and a feeble and rapid pulse. In short, the delayed operation is used only for those patients who we believe will not stand even the simple operation of drainage. Such a patient should be placed in Fowler's position, have heat applied to the abdomen, be given adequate doses of morphine by the clock, have Wangensteen suction applied to deflate the stomach and the intestine and be given liberal amounts of intravenous glucose and saline solutions. The operation should be undertaken as soon as the patient's condition warrants it, usually within a day or two.

The operation for appendicitis with peritonitis should involve as little trauma as possible. The peritoneum has usually been opened through a right rectus incision; the McBurney or gridiron incision has been used only in those cases in which it was felt the inflammatory process was lateral to the cecum. After the peritoneum is opened, the pus is removed by suction. No gauze is inserted into the peritoneal cavity. The old practice of inserting strips of gauze into the cavity for the purpose of walling off the infection was abandoned many years ago at the Children's Hospital. It is believed that this so-called "walling off" was a misnomer, and that the insertion of gauze into the peritoneal cavity defeated the very purpose for which it was used and resulted in spreading the infection and increasing the toxemia by traumatizing the serous surfaces of the bowel and abdominal wall. The question of the desirability of removing the appendix at this operation depends on the ability of the surgeon, the condition of the patient and the availability of the appendix. If it cannot be removed without a prolonged operation, involving trauma and hemorrhage, it is probably better to leave it in for removal at a later date. The practice of sewing the incisions up without drainage, as recommended by Giertz,[7] does not impress me favorably from the point of view of logic or of the results reported.

There is still considerable variance of opinion about drainage. Our usual practice is to insert one drain into the bottom of the pelvis and one into the iliac fossa. The drains are left in until the temperature approaches normal — usually after about a week, occasionally longer. The postoperative care is important. The patient is placed in Fowler's position, and parenteral fluids and morphine are used as a routine. If there is any tendency to postoperative vomiting or distention, Wangensteen suction is instituted at once. It is much easier to prevent ileus from increasing in its early stages than to cure it after it has become pronounced. Recently we have supplemented gastric or duodenal drainage by the oxygen tent with a high concentration of 90 to 95 per cent oxygen, as recommended by Fine, Hermanson and Frehling.[8] This appears to be very successful in deflating the bowel, and I believe will prove to be a very helpful addition to our therapeutic armamentarium against ileus.

Comparison of statistics of the results of the treatment of peritonitis is unsatisfactory and at best only suggestive, for the reason that what one surgeon may call general peritonitis, another may call spreading, and a third diffuse; and no surgeon really knows how diffuse the process is unless he has done a very improper operation. However, it seems to be the consensus that the mortality in children is higher than in adults. Our statistics at the Children's Hospital are better than most of those reported in adults, and better than any I have seen reported for children. In the last 632 acute cases, including the cases with peritonitis and the sickest patients, there was a mortality of not more than 3.5 per cent. In the last 204 cases in which there was gross rupture of the appendix and various degrees of peritonitis, including the most diffuse infection, the mortality was 7.3 per cent.

CONCLUSIONS

Too many children die from the complications of appendicitis.

The obvious solution to this situation is early diagnosis and early operation, which limits the problem to one of acute appendicitis without complications. In this stage of the disease the mortality is negligible.

The unwise use of cathartics in the presence

of abdominal pain is an important factor in causing peritonitis to result from appendicitis and in keeping up a high mortality.

Delayed operation or the Ochsner treatment is probably less effective in the child than in the adult but is occasionally desirable, even in the child. Its employment should be limited to surgeons of experience. When incorrectly used and misapplied, it is harmful, not helpful.

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THE CHAIRMAN: A few years ago it was my privilege to be in Baltimore and to hear a very excellent discussion of the problem of drainage in cases of appendicitis. It impressed me a great deal, and I learned much from it. We promptly got in touch with the author of this paper, who is professor of surgery at the University of Maryland School of Medicine, and he very kindly consented to come and talk to us about appendicitis as he has seen it: Dr. Arthur M. Shipley.

THE TREATMENT OF PERITONITIS COMPLICATING APPENDICITIS

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BALTIMORE

IN THE treatment of peritonitis complicating appendicitis there are many things to consider, but the two questions that are begging for a correct answer are when to operate and when and how to drain. These questions are answered in exactly opposite fashion by surgeons equally well qualified to do so, and this wide difference of opinion and practice is creating a great deal of confusion in the minds of physicians and laymen. Meanwhile the death rate from appendicitis is steadily mounting, and the mortality figures in perforated appendicitis vary in the different reports between 5 and 40 per cent. Much of this difference, however, is due to individual preferences in classification.

It is not possible to answer wisely either of the above questions in a dogmatic manner. Each patient is a separate problem, and while certain principles may be followed, there is no better way to keep the death rate high than to attempt to treat every case in conformity with some preconceived plan. The treatment of acute appendicitis, in which peritonitis may or may not be present, is probably the most serious problem that confronts the operating surgeon today, because the condition is so common and because there is so much difference of opinion as to whether to operate at once or to wait.

Early appendicitis, while the appendix is still in the acute stage, with no peritonitis, and chronic or recurrent appendicitis are in the main simple problems, if the surgeon has a chance to decide. There should be little difference of opinion as to what to do, and the mortality rate after operation should be almost negligible. In these groups of cases, after the diagnosis is made there is nothing to be gained by procrastination, except to increase the number of patients coming to operation later on with peritonitis. Over and over again, patients who have been amply warned by a number of previous attacks are admitted with peritonitis when the opportunity for a safe operative procedure is hopelessly lost.

The problem that concerns us in this paper, however, is peritonitis, and there are many factors requiring consideration. The time factor is the most important one. This determines very largely whether the infection is still confined chiefly to the appendix or has spread to the peritoneal cavity. At this point discussion may be simplified if some classification is adopted. A simple one is as follows.

We may classify acute appendicitis as catarrhal, suppurative, thrombotic, gangrenous or perforative. The term "suppurative" may be deceptive. What is meant is that the lumen of the appendix contains pus, but there is no perforation. The use of the word often causes misunderstanding, unless it is clearly stated whether we are speaking of the appendix or of the peritoneum. It is difficult not to include the term in speaking of the appendix, because very often, at operation, the appendix is found with its lumen filled with pus, and if care is taken it may be removed without rupture.

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The pathologist usually reports such a specimen as suppurative appendicitis. This diagnosis may confuse classification.

The real difficulty and much of the lack of uniformity in classification have to do with peritonitis. Many physicians think of peritonitis as being present only after perforation occurs. In many instances the peritoneal cavity will contain varying quantities of seropurulent matter, either localized or diffuse, complicating thrombotic, gangrenous or suppurative appendicitis before actual perforation occurs. In this paper we call this condition early peritonitis. After gross infection takes place, following perforation, we speak of the peritonitis as late.

When the appendix has perforated the peritonitis may remain localized, if the appendix is closely confined, or it may spread slowly or rapidly, depending largely on the size of the perforation and the kind, number and virulence of microorganisms present. If the surgeon elects to treat a late, diffuse peritonitis by waiting, the infection may subside and the peritonitis again become localized. Hence the confusion of terms — "early," "late," "localized," "spreading," "diffuse" and "subsiding" — in speaking of peritonitis.

Once the diagnosis of acute appendicitis is made, it is most difficult to say, before operation, just what has happened within the abdomen. Usually one of three conditions will be found so far as the peritoneum is concerned. There may be no peritonitis present; or there may be a seropurulent peritonitis associated with thrombotic, gangrenous or suppurative appendicitis, but with no gross soiling of the peritoneum by perforation; or there may be a perforated appendix with a heavily infected peritoneum. Occasionally there is a small, early perforation, partially sealed off, and the peritoneal reaction is that of early peritonitis. There were 13 such cases in the series reported below. They were treated as early peritonitis, without drainage of the peritoneal cavity, but the abdominal wall was drained.

Late diffuse peritonitis is a very grave and death-dealing condition. Here, several days have elapsed since the beginning of the peritonitis, and the suppuration has not walled itself off. The patient is anxious, hollow-eyed and dehydrated, the pulse is quick, and the leukocyte count and temperature are usually elevated. The abdomen is rigid, more or less distended, tender and painful. Paralytic ileus is present, so that enemas have been unsatisfactory and vomiting may have disturbed the acid-base balance of the body metabolism, with a sharp lowering of the chloride content of the blood.

At operation the intestines are edematous and have lost their smooth, shiny appearance. The small intestines are distended. Pus is widespread in its distribution, with many attempts at walling-off, so that coils of intestine are everywhere adherent, and when they are separated numerous pockets are found. Many of these patients succumb from the widespread infection, but some of them improve after drainage, only to die of obstruction later on.

If we could always be certain that the patient has acute appendicitis, and having decided that, could be sure that the appendix has not yet perforated, or if perforation has occurred, just what type of peritonitis is present, we should be better able to decide just what to do. Most of the reports dealing with the diagnosis and treatment of acute appendicitis are based on the operative findings, and pay little attention to the extremely difficult problem of deciding exactly what stage in the above classification the appendix and peritoneum may have reached when the decision as to treatment has to be made.

There are many things that make the time factor in determining the degree of peritonitis not entirely dependable. If a fecolith is impacted in the appendix, necrosis and perforation are apt to be hastened. Obesity often increases the difficulty. A deep-seated appendix overlaid by a thick abdominal wall and surrounded by masses of intra-abdominal fat may make diagnosis difficult and obscure the localizing signs. In children the whole progress from first symptom to perforation is apt to be more rapid than in adults. Whether previous attacks of appendicitis alter the time factor is a moot point. In spite of these variables it is often customary to speak of appendicitis as one-day, two-day, three-day, four-day, and so forth. These designations are often helpful, but may be misleading. It is widely agreed that patients taking cathartics are likely to have an earlier peritonitis, and that the rate of progress is directly in proportion to the number, strength and size of dosage taken.

The cecum may be high or low, and, dependent upon the length of its mesentery, fixed or mobile; the appendix may be long or short, the point of maximum damage may be at any point along its length, and the tip of the appendix may be found anywhere along the circumference of a circle whose center is the cecum and whose diameter is determined by the length and tortuousness of the appendix. The appendix may be free in the peritoneal cavity, entirely behind it, or have part of its length within the peritoneum and part outside it.

After the abdomen is open it should be decided at once whether the peritonitis is early or late. We classify the peritonitis as early — either localized, spreading or diffuse — if there is a quantity of sero-

purulent matter present, if the peritoneal covering of the small intestines is still shiny and not edematous, if there is no solid exudate except on the appendix itself, and if there is no walling off and no evidence of paralytic ileus. In these early cases we usually do not drain the peritoneum, provided a definite operative technic, presently to be described, may be carried out.

If, on the other hand, the peritoneum is edematous and has lost its glossy appearance, if solid exudate is present with walling off of pockets of pus, and if there are adhesions between the various abdominal structures, the peritonitis is considered late and is always drained, whether we are dealing with a late, diffuse peritonitis or a localized abscess.

Perforation is on the borderline between early peritonitis, in which the appendix may be removed without drainage of the peritoneum, and late peritonitis, in which drainage should be done. Before perforation takes place in acute thrombotic, suppurative or gangrenous appendicitis, there is in many cases a considerable amount of seropurulent exudate, and this may have spread beyond the immediate neighborhood of the diseased appendix. In our experience it is not necessary to drain the peritoneum. Because the abdominal wall is not so able to care for itself in the presence of infection as is the peritoneum, we sometimes drain it after closing the peritoneum, especially if much fat is present.

These and the subsequent statements are based on the behavior of 1106 cases of appendicitis treated on my service at the University Hospital, Baltimore, since June, 1930. Appendectomies done in the course of some other operation are not included.

There were 479 cases of chronic and recurrent appendicitis, with 2 deaths; 354 cases of acute appendicitis but no peritoneal involvement, with 2 deaths; 149 cases of thrombotic, gangrenous or suppurative appendicitis with early peritonitis, in which no drains were used, with 1 death; 28 cases of early peritonitis, in which the peritoneum was closed but the abdominal wall drained, with no deaths; 4 cases of early peritonitis, which were drained because of uncertainty as to a safe closure of the stump of the appendix, with no deaths; 41 cases of late, localized peritonitis or abscess, all of which were drained, with 1 death; and 49 cases of late, diffuse peritonitis, all drained freely, with 18 deaths. Altogether there were 833 patients without peritonitis, with 4 deaths, a mortality of 0.47 per cent, and 354 patients with some form of peritoneal involvement, with 20 deaths, a mortality of 5.6 per cent.

A brief analysis of these 24 deaths may be of interest. Of the 4 deaths in cases without peritonitis, 1 was caused by the anesthetic during an operation for acute appendicitis; the patient had a chronic rheumatic heart, but was considered a good risk as his heart was well compensated. One death was due to pulmonary embolism and 2 to pneumonia.

The actual causes of death in peritonitis may be divided into three chief classes. In one, death was the direct result of the peritonitis itself: paralytic ileus, dehydration, broken-down incision, toxemia and exhaustion. In another, the cause was some complication outside the abdomen: pneumonia, infarction, empyema, septicemia, and so forth. In the third class, death was due to mechanical obstruction. Of the 20 deaths in patients with peritonitis, 6 were due to intestinal obstruction. The only death in the 41 cases with late, localized peritonitis was caused by obstruction. This patient was admitted to the service ten days after the onset of abdominal pain and three days after the symptoms of obstruction had begun. At operation the abscess was well walled off and not large, but there was complete obstruction of a coil of small intestine incorporated in the abscess wall.

We use the McBurney incision if possible, because it has a number of advantages. It is made close to the anterosuperior spine, and high or low depending on the build of the patient and the location of maximum tenderness. After the peritoneum is opened every effort is made to localize the operation, as little packing as possible is used, and great care is taken not to traumatize tissues already damaged by infection. If the cecum is mobile it is gently rolled out and held by an assistant and the appendix is delivered. If the cecum has a short mesentery it may be possible to expose and deal with the appendix working inside the abdominal cavity, and if the appendix is postcecal or tightly adherent it may be necessary to free the cecum by incising the lower portion of the lateral gutter, thus making it possible to roll the cecum toward the midline.

"Whatever the condition found, an unhurried and painstaking attempt is made to free the appendix and secure the mesentery. Care is taken to handle the appendix with the fingers as little as possible. As soon as it is free it is wrapped in gauze, delivered as well as it can be, without too much tension, and removed." There are a number of ways to treat the stump. If possible, it is tied with fine catgut and turned in by a purse string of fine silk, with care to penetrate the lumen of the cecum no farther than necessary with the point of the needle. All raw surfaces are covered and the field of operation is kept clean with a sucker, rather than by sponging.

There is some anxiety in the minds of surgeons as to the possibility of infection in the wall of the cecum between the two ligatures. I saw one such case when I was house officer, but have not had this mishap in my own experience. Sometimes the appendix and cecum are very edematous and it is not wise to attempt to invaginate the stump of the appendix. It is tied with fine gut and may be covered with a patch from the mesenteric-like structure that often extends from the free surface of the terminal ileum to the cecum or a fat tag from the cecal wall. If such material is not available the stump is dropped back uncovered.

If the peritonitis is an early one the sucker is gently introduced into the pelvis and into the lateral gutter of the ascending colon, and any fluid in these spaces is removed. Care is taken to get a dry operative field and the peritoneum is accurately closed with fine chromic gut. A small drain may be left down to the peritoneum and the abdominal wall closed around it. Sutures are widely placed and very loosely tied.

One's desire to use little drainage material in the lower abdomen is based on a number of reasons, but the chief anxiety has to do with intestinal obstruction and postoperative adhesions. After any abdominal operation the possibility of ileus is a serious matter. Differentiation of paralytic ileus and mechanical obstruction is sometimes very difficult. To operate for paralytic ileus is still further to harm small intestines already dangerously distended, and to fail to operate promptly on mechanical obstruction is to invite disaster. The patient, his relatives, the surgeon and house officers go through an uneasy period until the issue is settled. It is true that morphine without atropine, decompression of the upper intestinal tract by gastric suction, and intravenous salt and glucose with decompression of the lower colon with the enema tube have markedly lessened the degree of paralytic ileus, but there still remains mechanical obstruction which may result from drainage. So we drain the peritoneum as little as our experience warrants.

Perforation is accepted by most surgeons as on the borderline between mild and severe peritonitis, but the issue is determined by the kind, number and virulence of the bacteria and the resistance of the host. Recently considerable attention has been paid to the kind of micro-organisms present. Study of the bacteriological reports reveals a wide divergence in the kind and number of organisms recovered from the unruptured, acutely inflamed appendix and from the pus of peritonitis complicating appendicitis. There are fairly uniform reports as to the aerobes, *Escherichia coli* (*Bacillus coli*) and various strains of streptococci being found in a high percentage of cases. Other aerobes are found as well; the staphylococcus, a gram-positive bacillus, and others. Occasionally *Hemophilus influenzae* (*B. influenzae*) is present.

Altemeier in 100 cases of perforated appendicitis recovered 16 different aerobes from the purulent material. Bower and his associates, reporting the flora in 55 patients, are in agreement so far as the aerobes are concerned.

There are a number of reports as to the presence of anerobes. Altemeier and Bower et al. differ sharply as to the presence of *Clostridium welchii* (*B. aerogenes capsulatus, B. perfringens, B. welchii*), but other observers are in accord with the latter in reporting a high incidence of clostridia, especially *Cl. welchii*. Another clostridium occasionally present is *Cl. oedematis maligni* (*Vibrion septique, Cl. septique,* bacillus of malignant edema). Altemeier reports for the first time the presence of *B. melanogenicum* in 92 per cent of patients examined. Bower et al. also report that 69 per cent of patients suffering from or recovering from spreading peritonitis had demonstrable and significant amounts of circulating antitoxin to *Cl. welchii*. Jennings found *Cl. welchii* in the lumen of the appendix in 90 of his cases.

Altogether, these bacteriological studies of pus in peritonitis, complicating the perforated appendix, are very disturbing to the surgeon. Most operators formerly believed that they were dealing with *Esch. coli,* different strains of streptococci and an occasional staphylococcus, and many suspected that *H. influenzae* played an unknown role in peritonitis and appendicitis.

But the knowledge that anaerobes are commonly present adds another factor that demands consideration. Under certain conditions, parasites attacking the tissues of the body prepare a pabulum in which the saprophytes flourish, and in addition to this the question of symbiosis demands immediate consideration. This is pertinent when it is recalled that progressive gangrenous ulceration of the abdominal wall is found in association with drainage tracts following perforated appendicitis, especially when it is remembered that this is a symbiotic infection between specific strains of the staphylococcus and streptococcus.

Meleney has stressed the possibility of symbiosis as an explanation of the varying behavior of suppurative peritonitis: "In any specific prophylactic or active treatment for peritonitis one must take into account the symbiosis of the commonest organisms found in the peritoneal exudate, namely *Esch. coli,* the green streptococcus and *Cl. welchii.*"

Late peritonitis is classified as localized, diffuse or subsiding. It is in the treatment of these groups

that there is so much difference among surgeons today. Late localized peritonitis, or abscess, may develop in a number of ways. Sometimes the peritonitis remains localized from the beginning. If the appendix is hedged in it may go on to perforation without producing a widespread infection. In these cases the diagnosis may be incorrectly made until the localized mass is apparent. This is very apt to be true if the perforated area of the appendix is away from McBurney's point: in the pelvis, high up on the right side, near the midline or postcecal.

Sometimes localization follows the waiting treatment of late, diffuse peritonitis, and is the result aimed at in this method of therapy. During the process of localization the peritonitis is a subsiding one. If the patient gives a history that indicates peritonitis, associated with perforated appendicitis, and several days have passed and there are the signs and symptoms of advanced peritonitis, with a hard, distended abdomen, vomiting, dehydration with acid-base imbalance, fever and rapid pulse, the outlook for recovery is bad. Most of the mortality in appendicitis is found in this group. The outlook is especially unfavorable in a person past middle age who is fat, with high diaphragm and little fighting reserve left in his tissues, especially if his heart and kidneys are already damaged. Whether to operate at once or to wait for improvement by otherwise treating these patients is the chief point of controversy among surgeons, so far as appendicitis is concerned.

The reported results are apparently in favor of the smaller group who postpone operation in these cases, and I am a convert to this method. There are a number of excellent papers in the literature supporting this view but there are also excellent reports advocating immediate operation. During the waiting period the treatment is almost identical with the postoperative treatment of peritonitis: decompression of the stomach and upper small intestine by the Wangensteen technic, morphine (without atropine), as this drug has a tendency to lessen paralytic ileus, the application of heat or cold to the abdominal wall, the control of dehydration and acid-base imbalance by the intravenous introduction of large quantities of salt solution and glucose and the decompression of the colon by use of the enema tube. To attempt to whip up the damaged and distended intestines by systemic cathartics is a questionable procedure. It is not possible in a paper of this length to discuss the checks and balances that should control this treatment.

Some discussion as to the treatment of localized peritonitis has taken place. It has recently been suggested that such cases be not operated on at all, as most of them will subside. I believe that localized peritonitis should be drained as soon as localization seems well established. It is impossible to limit this period in terms of days. The important thing is to approach the mass from directly above, if possible, to open into it without contaminating the rest of the peritoneal cavity, to make no attempt to remove the appendix, unless it is accessible within the localized area, and to drain both the localized peritonitis and the abdominal wall freely.

Drainage and suturing of the abdominal wall depend on the type of peritonitis, the kind of abdomen and the security of the stump of the appendix. In a borderline case a small drain may be used and the abdominal wall closed around it. It is my experience that if the abdominal cavity requires only a narrow strip of rubber tissue for drainage, no drainage is required. When the peritonitis is late, however, drainage should be free. One of the advantages of the McBurney approach has to do with the small need for suturing the different layers of the abdominal wall. Unless the drainage material is bulky and left in position for a long time, the wound edges will fall together as infection subsides and the drains are removed. There is little danger of evisceration following operation or of a ventral hernia later on.

Small, soft rubber tubing or Penrose drains make satisfactory material for drainage. They may be used together to advantage. Hard or large tubing is apt to produce necrosis; bulky drainage increases pressure and irritation, and cigarette drains, unless very loosely filled, are apt to act as stops rather than as an outlet for pus.

In the placing of drains the McBurney incision gives easy access to the three areas that often require drainage: the pericecal region, the right lateral gutter and the pelvis.

Much of the necrosis in the abdominal wall may be due to too many or too tight sutures. When it seems wise to drain the abdominal wall only, thin, long strips of vaseline gauze laid between the layers are useful.

Usually the same organisms are found about the appendix after perforation as are found normally within the cecum and appendix. The nearer the perforation is to the cecum and the larger it is, the greater the number and variety of organisms present, and the longer the perforation has existed, the more uncertain and complex the reaction between infection and tissues is apt to be. If only *Esch. coli* is present the outlook is fair; if the streptococcus is also present there is likely to be a greater reaction; and if *Cl. welchii* plays a part in the infection the outlook is still further complicated. There is abundant evidence that in all

forms of peritonitis complicating appendicitis the mortality rises with increase in the number and kind of organisms present.

Acute appendicitis presents itself as a somewhat different problem to the patient, to the practicing physician and to the surgeon. When a person has acute pain in the abdomen that is prolonged for any considerable period, he should not take a cathartic; he should take no food and should see his physician at once.

When a practicing physician sees a patient with abdominal pain and he believes that he is dealing with appendicitis he should send such a patient to a surgeon. It is a mistake to be influenced by the very considerable mass of literature that might lead him to wait. Osler said long ago, "There is no medical treatment for appendicitis." It is perfectly true that many cases of acute appendicitis will subside, if treated by rest, with nothing given by mouth, no catharsis, and an ice cap on the abdomen, but the mortality is confined almost entirely to the cases that do not subside.

It is also true that a localized mass following an attack of acute appendicitis may disappear without operation, but again, quite a number of them go on to suppuration and disaster. If all cases of appendicitis were early ones when they came to the surgeon, the problems and risks of peritonitis and drainage would largely disappear.

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Discussion

DR. JOHN M. BIRNIE, Springfield: I have been very pleased with this series of papers today, particularly as they all more or less deal with one subject, that is, immediate or delayed operation in cases of appendicitis. I have been extremely shocked and disturbed at the state of mind of a good many of the interns that come to the Springfield Hospital. These are high-class men, and a good many of them come from Boston; that is, they have received their medical and surgical instruction in Boston. I am tremendously surprised that these men seem to think that in all cases of acute appendicitis there are two courses open: you can operate at once or you can use the deferred treatment, the Ochsner treatment or whatever you wish to call it. Something is radically wrong with the teaching of today if medical students are sent out with the idea that in these cases of appendicitis it is perfectly safe to wait. It is absolutely wrong, and these three papers show that point clearly. If you sum up these papers, the indications for delayed treatment are in the cases that are so sick that you do not dare do anything else. In giving their indications for delayed treatment all three of these men have said that it should be carried out by only the most experienced surgeon with every facility at hand. It is not a preferred treatment.

Again, in Dr. Shipley's paper, if you noticed the way he expressed his opinion, it is a question of the very nicest decision based on years of experience. He has had unusual success. You cannot dispute the figures. They are wonderful. But if his teachings were sent out to the average surgeon the mortality would rise, I am quite sure. His judgment and his experience enable him to decide between very minute differences in the type of appendicitis with which he is dealing.

Now my figures, as few as they are, are based on about 200 cases. A short time ago I took out all the records of the severe cases of appendicitis in the Springfield Hospital. I wanted to get the different types of appendicitis, but I soon found in looking at them that I could not do it because of the difference in nomenclature. You could not tell with what you were dealing. Finally I came down to the point of checking back the cases in which the operating surgeon deemed it advisable to drain; there were 200 cases. Now of course these statistics do not mean very much, but one thing does show up: in all these cases there was, so far as we know, no case of intestinal obstruction — surely none during hospitalization. Hence, the dangers of certain types of drainage are not too great.

There has been some criticism among the men here for not sending cases to the surgeon early enough. I think their statements somewhat unfair because in this series of

200 cases that required drainage 40 per cent had been sick only twenty-four hours or less. It is something in the organism or something in the individual that determines the mortality and not necessarily the element of time.

In this same series, it was of interest to me that about 100 of the cases were between the ages of fifteen and fifty, and in those 100 cases there were only 2 deaths. Your mortality comes in the young and in those beyond the age of fifty. One third of all the cases were under fifteen, so that this group would come possibly into the same age classification as that of Dr. Ladd's cases.

The thought which I wish to leave in your minds, if nothing else, and which I think was brought out by these three papers we have heard, is that delayed operation in appendicitis is not warranted in any case unless the patient is so sick that you feel absolutely sure that any kind of intervention will push him over the brink.

Dr. James C. McCann, Worcester: Alert students in large centers, working with a wealth of clinical material, guide us by such valuable contributions as those of Drs. Walker, Ladd and Shipley. However, until we have unraveled the whole problem of peritoneal physiology, many will continue to resort to rubber-tissue drainage of the peritoneal cavity for local or diffuse peritonitis from acute appendicitis.

The attendant morbidity from the use of drains brings up difficult problems. The first problem, intra-abdominal morbidity from adhesions involving the small bowel, may be minimized by careful lateral placement of the drain into the coloparietal gutter. Much can be done for the second difficult problem, incisional morbidity resulting from infection, partial disruption and late hernia formation.

A year ago I began to use a revised routine in closing these incisions, based on several isolated procedures used by other men for varying purposes. It is with trepidation that I present a small series of 29 cases accumulated in this short period. But comparison of this group with a comparable group of consecutive cases handled by direct drainage through the operative incision is so striking as to warrant presentation of the routine for your consideration. There are three principles involved; compartmental drainage of the incision; lateral drainage of the peritoneal cavity; infrared therapy of the incision.

The first principle, compartmental drainage, is based on the fact that an incision breaks the fused anatomic continuity of tissue planes, converting potential into actual tissue spaces containing serum, some blood and bacteria — an ideal situation for infection. Two such compartments are established by the popular transrectus incisions for appendicitis: one within the rectus sheath resulting from lateral mobilization of the rectus muscle; the second a subcutaneous compartment between the rectus sheath and the mobilized subcutaneous fat. Two separate wicks of rubber tissue are so placed as to drain these compartments for six or seven days, when the cycle of infection, liquefaction and drainage should have been completed.

The second principle is lateral drainage of the peritoneal cavity, using a stab wound through the oblique muscle layers lateral to the rectus sheath. This eliminates the continuing contamination of the anatomic compartments of the incision that result from the common practice of placing a drain in the lower angle of the operative incision. It also gives an unbroken closure of the peritoneum.

The third principle, infrared therapy for the incision, is based on a report by Rankin and Graham.* They stated: "A decided improvement in the healing of these wounds has been noted during the past two years since we adopted exposure of the wound routinely to infra-red rays, commencing twenty-four hours after operation and continuing its daily use for seven to ten days." Such an observation by an authority like Rankin seemed worthy of trial in the management of contaminated and potentially infected incisions for acute appendicitis with peritonitis.

With the old routine of direct drainage through the incision, there were discharged under sixteen days only 10 patients or 30 per cent; with the special routine 20 patients or 60 per cent were discharged within the same time interval. Under twenty-one days with the old routine only 15 patients or 52 per cent could be discharged; with the special routine 25 patients or 86 per cent left the hospital. There had to be held from twenty-one to thirty-one days, 9 patients or 31 per cent under the old method, but only 2 patients or 7 per cent with the special routine. There had to be held from thirty-one to forty-one days under the old routine 4 patients or 14 per cent; under the special routine only 2 patients or 7 per cent. Grouping the last percentages, 48 per cent of those treated with the old routine had to be held for varying periods over twenty days whereas only 14 per cent of those under the special routine remained for similar periods of time.

There was a total group period of hospitalization of 645 days with the old routine and only 487 days with the special routine, a saving of 158 days or 25 per cent; this saving amounted to 5.5 hospital days per patient. But equally important is the preservation of the integrity of the incision under the special routine, so that theoretically such results should produce an extreme reduction in the incidence of postoperative ventral hernia.

Dr. Walker (closing): I do not want anyone to carry away an impression other than that our work in this deferred type of surgery was simply a clinical study from which there could be no conclusion. An individual with subacute appendicitis, if he is in condition to stand operation, certainly should be operated on. On the other hand, with apparent generalized peritonitis in an individual desperately ill, it seems preferable to treat him for toxemia for a certain period of time before operating.

The Chairman: If the next speaker arrived at this meeting early he could not have helped hearing his name mentioned a number of times in the papers which have been read on diffuse peritonitis, and I believe if there is any man's name in this country that is known to every surgeon and house officer in hospitals it is his. It gives me a great deal of pleasure to introduce to you the professor of surgery at the University of Minnesota Medical School: Dr. Owen H. Wangensteen.

*Rankin, F. W., and Graham, A. S. Closure of colostomy. Surg., Gynec. & Obst. 64:59-62, 1937.

ACUTE BOWEL OBSTRUCTION: ITS RECOGNITION AND MANAGEMENT

OWEN H. WANGENSTEEN, M.D.*

MINNEAPOLIS

IT IS a great honor of which I am highly appreciative that you should have asked me to come so great a distance to your annual meeting to discuss with you the problem of bowel obstruction. The voice of Reginald Fitz, who helped to clarify so many acute abdominal syndromes, still echoes to us from the past. In an address before the first meeting of the Congress of American Physicians and Surgeons fifty years ago, Fitz said:

> He [the physician] is to remember that in the light of exact knowledge nearly all cases of acute mechanical, intestinal obstruction die, unless relieved by surgical interference. That curative medical treatment has proven of sure avail only in a limited number of cases of intussusception, possibly a few of twist in the large intestine, and in certain cases of gallstones in the small intestine. That his first duty, after relieving pain, is to determine the capacity of the large intestine. That this is best accomplished during the first two days following the initial pain, before tympany makes the task more difficult, and pathological changes cause it to be more dangerous. Finally, that the means employed for this purpose represent the most efficient curative agent in his control. . . . In conclusion, acute intestinal obstruction is diagnosticated by exclusion. Its seat is fixed by injection. Its variety is determined by its seat, the age, antecedents and symptoms of the patient. Its treatment is surgical, on or after the third day, if the symptoms are urgent and forced injections fail to relieve.

Fitz's paper summarized the best medical opinion on the subject of bowel occlusion fifty years ago, and although Nicholas Senn, who presented the surgical aspects of the problem at the same meeting, was somewhat more insistent that patients with bowel obstruction should be operated on, nevertheless he found the results of surgical treatment to be practically identical with those treated in the manner outlined by Fitz (69 per cent mortality). Abdominal surgery then was in its infancy; the battle with obstructionists to practical antisepsis and asepsis had barely been won, and the x-ray was unknown. The intervening years have witnessed a better understanding and also accomplishment as concerns the general problem of bowel obstruction. In the practical management of the problem, however, much remains to be desired.

Read at the annual meeting of the Massachusetts Medical Society, Boston, May 31, 1938.

From the Department of Surgery, University of Minnesota Medical School, Minneapolis. This investigation was supported by a grant from the research funds of the Graduate School of the University of Minnesota.

*Professor of surgery, University of Minnesota Medical School; surgeon-in-chief, University Hospital.

From Boston a number of fine papers have come within more recent years which have helped materially to bring more light and understanding to the problem of intestinal obstruction. Gamble and his associates taught us how the loss of sodium chloride by vomiting added demineralization to dehydration and brought about alkalosis in high intestinal obstruction. The observations of McIver and his associates established that gaseous distention after abdominal operation was largely due to swallowed air. The work of White and his associate, Fender, indicating that a high bowel obstruction in the dog was well tolerated if the regurgitated gastric and duodenal contents were led into a loop of the gut beyond the obstruction, helped to set aside the toxic absorption idea as the chief concern in bowel obstruction. More recently Fine and his associates have come forward with the useful suggestion that inhalations of high concentrations of oxygen help by diffusion to displace the gaseous nitrogen trapped in the lower reaches of the distended gut.

In an address before this society in 1860—the halfway mark of its present age—a venerable member of your society, whose memory medical men now revere everywhere, established a generalization concerning medical addresses which still merits serious consideration. In "Currents and Counter-Currents in Medical Science," Oliver Wendell Holmes warned against the "lecture-room literature of adjectives" and cautioned against placing a premium upon "use of adjectives rather than of nouns substantive." In so far as I am able, I shall try to keep this admonition of our medical poet and philosopher in mind.

RECOGNITION OF BOWEL OBSTRUCTION

Of all the abdominal colics, only intestinal obstruction is characterized by a pathognomonic finding. The audition with the stethoscope of recurrent intestinal borborygmi and noises at the acme of concurrent crampy, colicky pains of short duration establishes the presence of intestinal colic. To be certain, enterocolitis, food indiscretions and functional intestinal upsets may be accompanied by intestinal colic, but on the basis of such general symptoms as vomiting, distention, fever and diarrhea, these can usually be excluded without difficulty. All other acute abdominal seizures, such as appendicial, biliary and renal colics, are to be differentiated by the absence of intestinal colic.

Strangulating Obstructions

Simple interruption of intestinal continuity is not ordinarily accompanied by abdominal tenderness. If, however, the bowel is strangulated or has undergone torsion, bloody fluid escapes into the peritoneal cavity and the abdominal wall exhibits tenderness, except in intussusceptions, in which the transuded fluid may be retained beneath the normal ensheathing cylinder. The parietal peritoneum is probably the most sensitive of all the serous membranes, and the sudden exudation of fluid into the free peritoneal space causes tenderness and rigidity. The degree of demonstrable rebound tenderness attending such an occurrence depends on the nature of the irritant. The most rigid abdomens are observed in perforations of the stomach or duodenum when hydrochloric acid and pancreatic juice escape into the peritoneal space. Blood or plasma which makes its appearance in the peritoneal cavity in strangulating obstructions constitutes a lesser irritant and in consequence provokes less tenderness and rigidity.

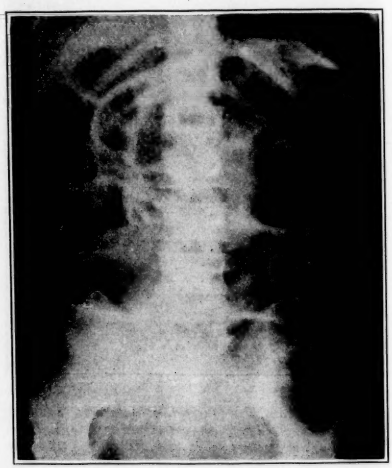


Figure 1. *Scout film of abdomen indicating a high grade of intestinal distention in a case of complete simple obstruction of the small intestine. Enterostomy done eight years ago; no recurrence of obstruction since. Today suction applied to an indwelling catheter would be tried in such a case first, care being taken to intubate the duodenum, the catheter being pushed well into the small bowel (Fig. 6).*

In primary inflammatory lesions which secondarily give rise to mechanical obstruction of the bowel, such as inflammation plus simple obstruction, the differential diagnosis of such a condition and a primary strangulating obstruction may be difficult or impossible.

The Value of X-Rays in Diagnosis

A single scout film of the abdomen made with the patient lying supine affords indispensable information in determining whether gaseous distention is present and in indicating what segments of bowel are distended. The visualization of gas in the small intestine of the adult is distinctly unusual, and ordinarily indicates intestinal stasis. In obstructions of the large bowel, however, the distention is usually confined to the colon by the competent proximal ileocolic valve and sphincter. Participation of the small intestine in the distention in such cases in the experience of the writer is unusual. Great colonic distentions of acute mechanical origin when unrelieved perforate frequently, and it is usually the cecum which gives way — the portion of the colon with the largest diameter. The stethoscope rather than the roentgen ray, however, makes the diagnosis. Roentgenologists should be wary of diagnosing mechanical bowel obstruction from scout films without knowledge of the clinical findings. It is to be remembered that in the film made with the patient lying supine, the diameter of the distended intestinal coils is usually enlarged 25 per cent. If the film is made with the patient prone, it shows the true diameter of the distended bowel. The great advantage of the film made with the patient lying supine, however, is that it permits tracing out the continuity of distended intestinal coils (Fig. 1). Though a film taken with the patient erect is of value in all suspected perforative lesions of the gastrointestinal canal, it does not lend much useful information in solving the problem of where the bowel is obstructed. It does lend a helpful impression, however, concerning the extent of fluid retained in the obstructed bowel, and is therefore distinctly useful in more chronic types of obstruction in which fluid retention is great. Under such circumstances, the gas in the bowel may be obscured by the fluid in the usual scout film taken in the supine position.

Is the Obstruction in the Large or the Small Bowel?

Vomiting, although a prominent symptom in obstruction of the small bowel, may be altogether absent in obstructions of the colon. Fecal vomiting is synonymous with obstruction of the small bowel. Reflex vomiting may occur in acute colonic obstruction, but unless the ileocolic valve and sphincter are incompetent — an unusual occurrence — vomiting is certainly not prominent. If a duodenal tube is passed into the stomach and

suction is applied in colonic obstructions there is frequently no return of fluid and only a little gas. In high-grade obstructions a copious return of stercoraceous fluid is usual, unless vomiting has immediately preceded intubation.

Visualization of the gaseous distention as indicated in the scout film of the abdomen is a helpful measure in determining whether the obstruction is in the colon or the small intestine. Occasionally, however, the evidence is inconclusive unless barium is administered by rectum.

Is the Obstruction Complete or Incomplete?

The enema is of little value in diagnosis, for the bowel beyond the obstruction is physiologically as well as anatomically normal. However, patients frequently come to the hospital several days after the onset of their illness because gas and feces were expelled with enemas. Such is often the case in high complete occlusions or in incomplete obstructions anywhere in the intestinal canal.

Figure 2. *Roentgenogram in incomplete acute obstruction of the small bowel (old case). Gas is still visualized in the colon. The absence of abdominal tenderness indicated that the obstruction was simple in nature. Successfully decompressed by enterostomy without recurrence of obstruction; today such a patient would be best treated by suction.*

A large caliber of the distended coils of small intestine, with no gas in the colon after evacuant enemas,* indicates a complete obstruction of the small intestine. Lesser distention of the small bowel with persistence of gas in the colon signifies a partial obstruction (Fig. 2). In obstruction of the large bowel, the diameter of the distended colon is the best determinant of whether the occlusion is partial or complete.

*The writer is firmly convinced that enemas should be studiously avoided in all inflammatory intraperitoneal lesions. Who has not seen a localized suppurative perforation of the appendix become generalized following administration of an ill-advised enema?

WHAT ARE THE EFFECTS OF OBSTRUCTION?

Before considering the therapeutic measures which may be invoked to deal with acute intestinal obstruction, it is important to have in mind what the effects of obstruction are. Essentially these are of two kinds: the effect of the obstruction on the organism as a whole, and its effect on the bowel wall.

Figure 3. *Scout film in a patient with acute occlusion of the left colon. There was no vomiting; the small intestine did not participate in the distention. Operative decompression is indicated (Fig. 11).*

The general consequences of intestinal obstruction, save when the bowel wall itself has been badly damaged, are limited to two: first, dehydration and dechlorination, with corresponding blood chemical changes, in patients in whom copious and protracted vomiting has been a prominent feature; second, loss of blood in strangulating obstructions in which the length of the infarcted bowel is great. In such cases the blood loss may be large, as heralded by an increase in pulse rate and a low arterial blood pressure.

The effects of obstruction on the bowel wall

are in part obvious: the bowel distends with fluid and gas and the intraluminal pressure increases. When the sustained intraluminal pressure exceeds the venous pressure and approximates the capillary blood pressure, adequate oxygenation of the bowel wall becomes compromised, with attendant sequelae. Perforations in colonic obstruction occur not uncommonly; in the small bowel, perforation is decidedly uncommon, but as the permeability of the bowel wall increases with decreased blood flow and impaired viability, it loses its capacity to restrain transperitoneal migration of noxious substances. In strangulating obstructions, these deleterious effects on the bowel wall are observed earlier. The threat to the peritoneum in every strangulating obstruction is real.

In simple obstructions of the upper small intestine, regurgitant vomiting serves to relieve the tension of augmented intraluminal volume and increased pressure. In lower ileal obstructions, however, because the bowel is not emptied readily by vomiting, sustained increases of intraluminal tension maintained over long periods may permit transudation of injurious agents from the lumen of the bowel into the peritoneal cavity.

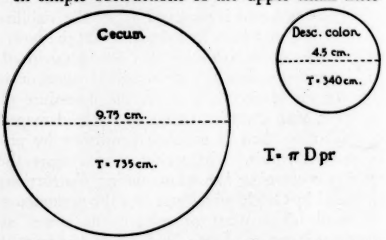


Figure 4. *The stress or tension (T) on the bowel wall in the cecum, where the diameter is large, and in the descending colon. The intraluminal pressure was 24 cm. of water (Fig. 3).*

Sustained intraluminal pressures of 4 to 18 cm. of water have been observed by the writer in instances of small bowel obstruction at operation. In the main, pressures over 10 to 14 cm. of water have been present only under unusual circumstances. In the colon, on the other hand, because gas and fluid may continue to be dumped into it from the proximal reaches of the small bowel, and because no regurgitation into the ileum can occur unless one of the lips of the ileocecal valve is deficient, high intraluminal pressures frequently attend acute colonic obstructions. The bursting-strength of a bowel wall which has been obstructed for forty-eight hours or more is decreased notably, and the tension on the wall in colonic obstructions therefore often exceeds its bursting-strength (Fig. 4).

The old adage that high obstructions are the most vicious is now outmoded in the knowledge of what saline solution can do to replace fluids and minerals lost by vomiting, and in the demonstration of the effects of tension on the increased permeability of the bowel. Low obstructions hold the greatest risk to life, for release of tension is the only agent which can affect the issue favorably.

Therapy

Saline Solution. In all high obstructions in which demineralization (loss of sodium chloride) and dehydration may occur, adequate replacement with liberal quantities of physiologic saline solution is in order. Oliguria attends all obstructions in which the fluid-loss item is great, and a free excretion of urine appears only when the dehydration and sodium-chloride loss have been repaired sufficiently to restore the plasma chlorides to their normal value. In practice, I have found that a satisfactory urinary excretion in which the presence of sodium chloride can be demonstrated (800 to 1000 cc. every twenty-four hours) is as reliable a test of restoration of the normal blood-chloride value as is its actual chemical determination. In the frequent colonic obstructions in which vomiting is absent, the generous administration of saline solution is in no sense a specific.

Blood Transfusions. The transfusion of blood in adequate amounts is urgently indicated in all strangulating obstructions in which quickening of the pulse suggests a significant blood loss into the strangulated segment. The transfusion of blood is in order, as well, in all simple obstructions in which a high intraluminal pressure has brought about considerable transudation of fluid from the portal blood stream. The latter group of patients, however, rarely exhibit significant decreases of systolic blood pressure.

Oxygen. Inhalations of high concentrations of oxygen, as suggested by Fine and his associates, should prove of value in controlling intestinal distention of whatever origin when suction applied to an indwelling duodenal tube is used simultaneously. By reduction of the percentage of nitrogen in the inspired air to a minimum, the nitrogen tension in the blood plasma is similarly decreased and the nitrogen in the bowel lumen, which ordinarily does not diffuse readily through the intestinal wall because of the high tension of nitrogen in the plasma, slowly diffuses into the venous blood of the portal system and is exhaled — a measure which will prove successful in direct ratio to the decrease of nitrogen tension in the plasma and the success with which swallowed increments of air are re-

moved by an inlying duodenal tube. In this connection, my associate, Dr. John R. Paine, and I in 1933 tried out the value of 60 per cent concentration of oxygen on a small series of cases with intestinal distention, the patients being maintained in oxygen tents. Roentgenologically we had difficulty in satisfying ourselves that a diminution in distention attended this use of oxygen. However, without the aid of suction which removes increments of swallowed air that contribute to the existing distention, even though the inhalation of high concentrations of oxygen causes nitrogen to

Suction apparatus used in certain cases of intestinal obstruction
University Hospital-University of Minnesota

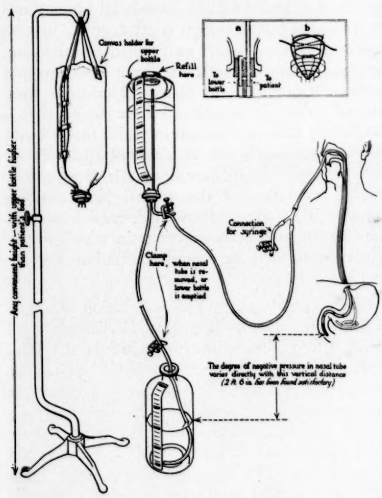

a
b
Canvas holder for upper bottle
Refill here
To lower bottle
To patient
Connection for syringe
Clamp here, when nasal tube is removed, or lower bottle is emptied
The degree of negative pressure in nasal tube varies directly with this vertical distance (2 ft. 6 in. for 6mm found satisfactory)

Figure 5. *Suction apparatus for treating intestinal distention. When the fluid returns are great in amount, a trap bottle should be included in the system to permit return of aspirated fluid by proctoclysis.* (Reproduced by courtesy of the *Journal of the American Medical Association* [*101*:1538, 1933].)

diffuse from the bowel wall, one only replaces one intestinal gas with another. When used in conjunction with suction, the method undoubtedly has real merit and should find wide usage. Fine has apparently been able to show that high concentrations of oxygen (95 per cent) can be given over long periods without harm. An obstacle to earlier use of the method was the belief that inhalation of high concentrations of oxygen caused desquamation of the bronchial epithelium and predisposed to pneumonia — a thesis to which most physiologists subscribed and which the experiments of Binger and his associates on oxygen poisoning supported. The amount of gas removed by this method will depend on the degree of lowering of the nitrogen tension in the blood plasma and the extent of the distended bowel. When extreme lengths of bowel are concerned, a greater quantity of blood with a low nitrogen tension is available for taking up nitrogen from the bowel. The method may therefore prove to have greater worth in inhibitive (paralytic) than in mechanical obstructions.

Strangulating Obstructions

All strangulating obstructions must of necessity be operated on as soon as the diagnosis is made. With the single exception of mesenteric thrombosis or embolism, all such obstructions may be treated as simple occlusions, granted that the strangulating agent is removed before the viability of the bowel has been impaired. Once the bowel is no longer viable that segment must be sacrificed. In the main, unless the strangulated segment is high or the subject is a child, the procedure of choice in such cases is to exteriorize the damaged bowel, rather than to establish continuity by primary anastomosis. The writer cannot share the opinion concerning late strangulating obstructions expressed by Gatch, who states that the peritoneum can wall off a piece of gangrenous bowel as readily as it can an abscess. Perforation of a loop of bowel that has lost its viability is so menacing that every effort should be bent to excise the loop before that dreaded event occurs. Whereas now and then a non-viable segment of bowel becomes isolated and walled off with formation of a bowel fistula, the far more frequent event is perforation into the free peritoneal space, with disastrous consequences.

Simple Obstructions

Suction. In simple obstructions the choice of a therapeutic agent is not easy. A number of obstructions, particularly simple adhesive occlusions, may be dealt with satisfactorily by suction applied to an indwelling duodenal tube. If one bears clearly in mind that all patients with bowel obstruction exhibiting signs of peritoneal irritation may have a strangulating obstructive mechanism and should be subjected to early operation, one is not likely to fall into the error of treating by conservative means a patient who should have operative intervention. Further, it must be remembered that all patients with acute colonic obstruction exhibiting great distention should be decompressed at once by operation because of the hazard of perforation.

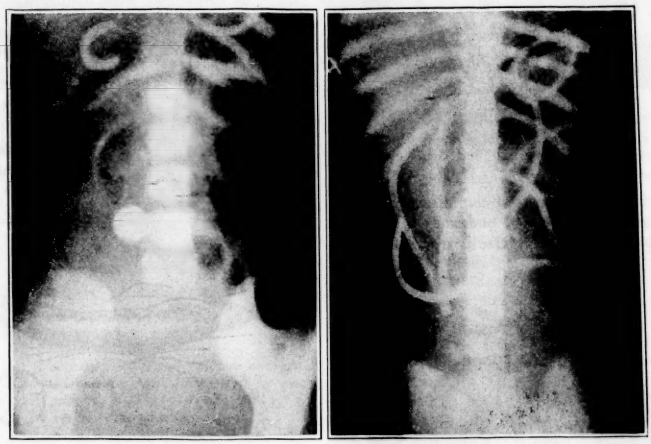
How long may one reasonably persist in attempts to decompress an obstructed small bowel by

suction without recourse to operative intervention? A recent paper by van Beuren and Smith on bowel obstruction suggests in the main that suction applied to an inlying duodenal tube is largely an ancillary mode of relieving intestinal distention, and that operation, even in cases deemed suitable for relief by suction, is the procedure of choice. This expression undoubtedly represents the opinion held currently by many surgeons in many places. Yet, since the efficacy of suction was demonstrated for the treatment of certain types of acute mechanical obstruction in 1931, there is probably not so large a group, — but, nevertheless, one which counts among its numbers many who have a wide experience and great interest in the obstruction problem, — who continue to insist that a fairly large number of mechanical obstructions of adhesive origin can be treated safely by suction applied to an inlying duodenal tube.

The suggestion of Abbott and Johnston of placing a small balloon, which may be inflated, at the tip of the duodenal tube to facilitate its transit into the lower reaches of the bowel will widen the scope of usefulness of conservative means of decompression. Yet it is not to be forgotten that the ordinary duodenal tube has for years been pushed into the lower ileum (Fig. 6*b*). Although the placement of a balloon at the end of the catheter makes it somewhat more difficult to intubate the duodenum, it is to be freely admitted that once the duodenum has been entered, distention of the bowel by the balloon will considerably enhance the rapidity with which the tube may be pushed into its lower reaches.

Clinical Lessons Taught by Suction

The wide usage which suction has enjoyed in the treatment of intestinal distention has been a means of adding valuable information to our knowledge of abdominal disease. It has taught that postoperative vomiting can be eliminated by keeping the stomach and duodenum empty, and that early postoperative mechanical obstructions have practically disappeared wherever suction has been employed after abdominal operations to prevent distention. It has been learned too, unfortunately, that patients may die of peritonitis despite absence of distention. The conservative management of many acute lesions of the abdomen has been widened by the knowledge that suction constitutes an effectual means of dealing with intestinal distention.



A B

Figure 6. *a. Fairly high-grade, simple obstruction of the small bowel. b. Nine hours after duodenal intubation the catheter was pushed into the lower reaches of the bowel. An inflatable balloon (Abbott) may be placed on the tip of the catheter to facilitate migration of the tube into the lower ileum.*

Choice of Operative Procedures for Obstruction of the Small Bowel

In simple obstructions the simplest operative procedure is the safest. There are, to be sure, errors of commission and omission into which the experi-

enced surgeon may fall in the management of bowel obstruction. In the main, however, in simple obstructions the errors of commission are more to be feared than those of omission; in strangulating obstructions, the reverse is true. The most serious bungling in the management of simple obstruction results from attempts to perform the operation of election when a simple decompressive operative procedure would suffice. If the preoperative criteria of physical examination suggest a simple obstruction and no bloody fluid is seen when the peritoneal cavity is opened, the surgeon will do well to content himself with the performance of enterostomy. It is to be fully conceded that enterostomy shares in a sense the shortcomings of suction applied to an indwelling tube: it is a "blind" procedure. Yet the surgeon cannot eviscerate and examine in a thorough manner all the distended bowel without pyramiding the risk. Further, he is often led on by this chase for adhesive obstructions to temptations which he can no longer resist, and too often finds himself involved in difficulties from which he cannot extricate himself without considerable risk to the patient — the most important person at an operation. The great danger is that the bowel may be opened in such a dissection.

Figure 7. *The technic of performance of a satisfactory aseptic enterostomy: segregating and "milking out" a distended loop.*

Figure 8. *The technic of performance of a satisfactory aseptic enterostomy: aspiration of residual air, and fluid.* (Reproduced by courtesy of the *Archives of Surgery* [26: 958, 1933].)

Figure 9. *The technic of performance of a satisfactory aseptic enterostomy: when a thinned-out bowel is deflated, its wall immediately gains substance and may be sutured safely.* (Reproduced by courtesy of the *Archives of Surgery* [26:958, 1933].)

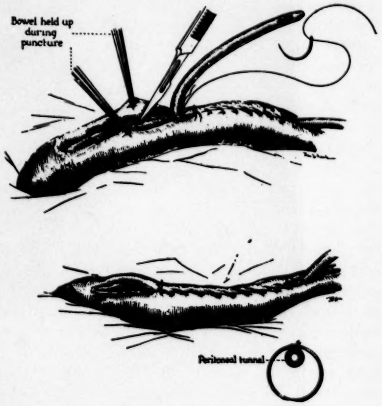


Figure 10. *The technic of performance of a satisfactory aseptic enterostomy: placement of catheter in the lumen of the bowel, with a resultant peritoneal tunnel, which will close spontaneously without leakage when the catheter is pulled out, granted that the continuity of the intestinal canal has become re-established.*

All operations for acute bowel obstruction must be done aseptically. Contamination or spillage is synonymous with failure. The sterility of the peritoneum must be preserved inviolate. In the experience of the writer, enterostomy when aseptically performed, despite the admitted weakness that it is an incomplete procedure, is nevertheless the safest in late, simple obstructions. To be sure, simple obstructive agencies such as a band, when visualized, should be cut. Enteroanastomosis is to be studiously avoided in acute obstructions. Even in chronic obstructions it not uncommonly gives rise to subsequent trouble, and is rarely indicated in obstructions of the small bowel, unless excision of the short-circuited segment is con-

templated. The surgeon's prayer in the surgical management of simple obstruction should be: "Lead us not into temptation" of doing too much.

Simple Acute Obstructions in the Colon

Simple acute obstructions in the colon are often vicious and hold out a serious threat to life. The preoperative scout film of the abdomen, when correlated with the clinical findings, generally affords

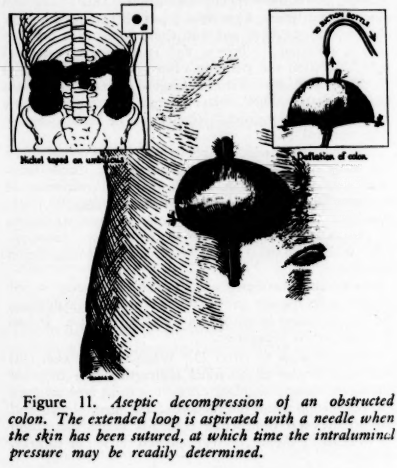


Figure 11. *Aseptic decompression of an obstructed colon. The extended loop is aspirated with a needle when the skin has been sutured, at which time the intraluminal pressure may be readily determined.*

so accurate an indication of where the obstruction may be found that it may be relied on as a safe guide. The more usual location is in the pelvic colon, and the most frequent obstructing agent has been found to be carcinoma. The occurrence of chronic volvulus and cases of neurogenic ileus, however, are to be borne in mind. Decompression of the transverse colon when the obstruction lies beyond it is the simplest operation, and one which can be done with minimal risk (Fig. 11). Cecostomy is not a good operation because it is difficult to open the cecum aseptically when distention is great.

SUMMARY

The diagnosis of acute bowel obstruction can usually be made without difficulty. By proper evaluation of the symptoms and findings, when these are related carefully to the disclosures of the scout film of the abdomen, one may usually decide with confidence whether obstruction is present, whether it concerns the large or small bowel, whether it is simple or strangulating in character, and whether it is complete or incomplete. Without recourse to operation, one cannot say precisely just how the bowel is obstructed, and even then it may be difficult.

In high obstructions, dehydration and demineralization may occur through the agency of vomiting. In all low obstructions, the hazard to life lies in the compromise of the viability and permeability of the bowel wall through the agency of increased intraluminal pressure. In strangulating obstructions, this danger is augmented because of the greater rapidity with which these changes occur. In addition there is the hazard of loss of blood.

The therapeutic agents of value in the management of acute bowel obstruction are as follows:

1. Replacement of fluid and electrolyte losses by liberal para-oral administrations of saline solution.
2. Transfusion of blood in strangulating obstructions.
3. Inhalation of high concentrations of oxygen (when used in combination with suction applied to an inlying duodenal tube) to encourage diffusion of gaseous nitrogen from the distended bowel.
4. Suction enjoys a wide range of usefulness in the management of all types of intestinal distention. A large number of simple obstructions, adhesive in character, can be satisfactorily managed by suction alone. It has also been a means of adding valuable information to our knowledge of acute abdominal disease and its management.
5. Operative decompression of the distended bowel or removal of the obstructing agent without peritoneal soiling before the bowel wall has become abnormally permeable, permitting the transperitoneal migration of noxious substances. The keynote of treatment in simple obstructions should be the release of intraluminal tension. In strangulating obstructions, the bowel, if found to be non-viable, must be excised.

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Discussion

Dr. Howard M. Clute, Boston: There is a tendency among physicians to feel that intestinal obstruction is primarily a surgical disease since the treatment is usually carried out by surgeons and that only a passing or cursory knowledge of intestinal obstruction is needed by the medical man. It is, however, generally recognized that the mortality from acute intestinal obstruction is directly related to the length of time that elapses before treatment is undertaken, and therefore, the ease and speed with which this diagnosis is made are of vital importance to the patient. All of us, therefore, who practice medicine must be deeply interested in the diagnosis of this condition. We are all greatly indebted to Dr. Wangensteen, who has made the clinical picture so clear and who has added so materially to our understanding of the methods of treatment.

I believe that some degree of intestinal obstruction is a complication of abdominal disease far more often than we realize, and that increasing knowledge of its typical symptomatology will add very greatly to our effectiveness in dealing with these patients. Dr. Wangensteen has emphasized here and elsewhere the significant clinical facts which must not be overlooked, and which point clearly to bowel obstruction. They are cramp-like, colicky intestinal pains, audible gurglings of gas and more or less obstipation. When necessary, the diagnosis may be confirmed by an x-ray plate of the abdomen.

These symptoms are precise and definite, and should demand immediate action — not delay and watchful waiting. It is, however, a very common tendency, particularly in postoperative abdominal cases, to attack this syndrome of colic and borborygmus by enemas, injections of Pituitrin and hot flaxseed poultices. In a great many hospitals in New England I have rarely found a duodenal drainage tube commonly used, and even more rarely have I seen constant Wangensteen suction employed. This method of managing obstruction and partial obstruction is so simple and so effective that there is no excuse for its limited use in smaller community hospitals. Nurses can be readily taught to pass a duodenal tube, and certainly all house officers should be very familiar with its use. Suction drainage is a simple procedure; yet the results obtained are so satisfactory that one wonders now how we ever practiced surgery before the Wangensteen method was devised.

One of the distinct problems that enters each case of complete or partial intestinal obstruction is the decision as to when one should give up the use of duodenal drainage and operate to relieve obstruction. No rules can settle this point, which surgical judgment must govern, but I emphasize the danger of repeatedly relieving an obstructed bowel by Wangensteen suction. This I have seen done several times. Operation is put off because duodenal drainage brings relief, but obstruction recurs each time the tube is removed. I believe that recurrence of intestinal colic, distention and gurgling a few hours after their relief by duodenal suction demands surgical intervention. I am satisfied that repeated delays in operating on such cases may so deplete the patient's vitality that he becomes a very poor surgical risk.

One must emphasize the points which Dr. Wangensteen has made regarding the differentiation of a simple intestinal obstruction and one complicated by involvement of the circulation of the bowel, that is, a strangulation obstruction. He says: "Intestinal colic without tenderness of the abdomen signifies simple obstruction. Intestinal colic with abdominal tenderness indicates strangulation obstruction."

In strangulation obstruction no delay in surgery is permissible once the salt and fluid balances are re-established. No reliance must be placed on duodenal suction. Immediate surgery is imperative.

Finally, I wish to stress Dr. Wangensteen's assertions that some degree of intestinal obstruction is a frequent finding in acute abdominal cases; that colic, borborygmus and obstipation mean obstruction of the small bowel; and that small-bowel obstruction demands immediate action for its relief by the use of Wangensteen suction or surgery, or both.

The Chairman: The final paper of the morning is on a subject which also has interested us very much. It is the question of non-inhalation anesthetics, about which there is a great difference of opinion, and about which all of us have much to learn. It seemed to us that we could do no better than to choose as our speaker one whose experience has been so great and in whose judgment we have so much confidence. I call on Dr. Lincoln Sise of the Lahey Clinic.

CLINICAL EVALUATION OF SOME NONVOLATILE ANESTHETIC DRUGS

LINCOLN F. SISE, M.D.*

BOSTON

THERE is a group of anesthetic drugs about whose value and usefulness and particularly about whose danger there is considerable uncertainty and difference of opinion among many physicians. It therefore seems fitting to review the situation and if possible to determine their position.

The group is a somewhat heterogeneous one and consists, first, of general anesthetics for rectal and intravenous use, and secondly, of locally acting drugs for spinal use.

GENERAL ANESTHETICS FOR RECTAL USE

Of this group tribromethanol (Avertin) is most commonly used; paraldehyde is used to a considerable extent; the sodium salt of n-methylcyclohexenylmethyl-barbituric acid (Evipal) is used to some extent; while sodium ethyl-l-methylbutyl-thiobarbiturate (Pentothal) is used very little. These drugs are introduced in solution into the rectum and are absorbed from there into the general circulation. They are eliminated for the most part in two stages. They are first detoxified in the liver by combining with glycuronic acid and are then excreted by the kidneys. Paraldehyde is excreted to some extent through the lungs but mainly directly by the kidneys. The depth of anesthesia is thus determined by the balance between absorption and elimination. Usually absorption is at first much more rapid than elimination. The amount of drug in the circulation increases rapidly, the patient is anesthetized, and the plane of anesthesia gradually deepens. As the amount of drug left in the rectum lessens, absorption slows, while elimination continues unabated. For a time the two run approximately parallel and a level plane of anesthesia is maintained. As the amount of drug remaining in the rectum is lowered still further, absorption decreases to such an extent that the balance is thrown to the other side. The depth of anesthesia then lessens and recovery begins.

All this, however, takes place without intervention by the anesthetist. Moreover, there are great variations in the character of the cycle. Absorption may be extremely rapid, a large amount of the drug may be rapidly thrown into the circulation, and the patient may be quickly anesthetized to marked depth; or absorption may be very slow and prolonged so that elimination is almost able to keep pace with it, and the patient is never fully anesthetized. Thus the result of a given dose is quite uncertain and is largely unpredictable.

Under the full effect of Avertin, respiration is quite uniformly depressed, blood pressure is lowered and the pulse is frequently unaltered or even slowed, while volume is well maintained. The pulse may, however, become rapid with decreased volume. This is an unfavorable development. On the addition of an inhalation anesthetic the blood pressure tends to rise toward the original level, and this is likely to be accentuated further by the reflex stimulation of operation. Evipal, at least in the doses usually recommended, appears to be less depressing than Avertin but also less effective. Paraldehyde is less depressing to respiration but tends to be accompanied by the formation of mucus.

It is evident that the anesthetist has little if any control over the depth of anesthesia beyond his choice of the original dose of the anesthetic. Washing out the rectum can of course be tried, but this is uncertain and usually impractical. Thus it behooves the anesthetist in choosing the dose to be sure that it is not too large. If a large dose should be selected and absorption should happen to be extremely rapid the patient might easily be plunged to too great a depth. The dose must always be on the safe side. While the fractional method of administration will help in determining the dose, nevertheless it should in practice always be smaller and the probable effect less than might otherwise be desirable. The balance of anesthesia must be made up in some other manner, preferably by some anesthetic with a high degree of control such as an inhalation anesthetic, especially one of the gases. Only in rare cases, and then only when the lightest plane of anesthesia suffices, as in painful dressings, should rectal anesthesia be used alone. Moreover, the action of these drugs is more that of hypnotics or narcotics than it is that of true anesthetics, and anesthesia actually begins only as the degree of depth approaches the danger point. Because of this and because of the almost complete lack of control, the policy should always be to play safe.

Read at the annual meeting of the Massachusetts Medical Society, Boston, May 31, 1938.

From the Department of Anesthesia, The Lahey Clinic, Boston.

*Anesthetist, The Lahey Clinic.

Besides this general attitude of caution due to lack of control, there may be more specific reasons for caution under certain conditions. If there is marked loss of function of liver or kidneys the process of elimination may be so slowed as to make these drugs dangerous. The lowering of blood pressure probably increases susceptibility to shock, so that it is best not to use them in situations where shock is likely to develop. Because of their depressing effect on respiration they should not be used when there may be difficulty in respiration, as in hydrothorax, distended abdomen or obstruction to respiration from any cause. It is particularly important to be on the watch for any possibility of obstruction to respiration. Because of the increased danger of cerebral anemia from the drop in blood pressure they should not be employed when there is greatly increased intracranial pressure.

The importance of anoxemia must be particularly stressed. This can easily occur at any time from depressed or obstructed respiration. Anoxemia increases the toxicity of these drugs for the liver, and probably lowers the functional capacity of this organ and hence slows elimination. It decreases cellular respiration, injures the entire organism and increases susceptibility to shock. If at all marked it further depresses respiration and may initiate a vicious circle of events similar to that in the postoperative period described later. Thus it is of the utmost importance to guard against it before, during and after operation.

I believe that close attention to the possibilities of anoxemia is one of the key points in the employment of these drugs. They have little direct harmful action of their own, but their possibilities for harm through the medium of anoxemia are almost unlimited.

In this connection I should like to call particular attention to the danger of the postoperative period. During the operative period the patient is under the direct observation of the anesthetist and presumably is guarded carefully from anoxemia. During the postoperative period he is not under such close or such competent observation and yet, owing to the slow recovery from this form of anesthesia, he may remain in deep narcosis for some time. This may therefore be the most dangerous time. Incidentally, I consider unwise the custom which prevails in many hospitals of darkening the patient's room during recovery even though he is still deeply narcotized. This prevents the early recognition of a slight shade of cyanosis, a matter which is of the utmost importance. The room should be kept light at least until the patient is definitely recovering consciousness.

During the recovery period, anoxemia may occur from one of two causes. First it may arise from simple lowering of respiration due to the depressing influence of these drugs. Vascular depression is usually also present and slows up cellular respiration still further. Great harm may be done the patient if this is allowed to continue for some time. It may become cumulative in its action. If anoxemia becomes of more than slight degree it tends to deepen the narcotic effect of the drug. This depresses respiration still further, and thus a self-perpetuating train of effects of growing intensity is set up, which if not broken somewhere may conceivably lead eventually to death.

The second cause for anoxemia during recovery is the occurrence of obstruction to respiration in addition to narcosis and depression of respiration. If the history of Avertin is read, particularly the history of its early use in Germany, one cannot help but be struck by the frequent mention of the term "secondary depression of respiration." A patient goes through operation successfully and is returned to bed in good condition, but several hours later respiration becomes depressed and he becomes cyanotic and dies. This sequence of events does not seem reasonable. If he goes through the early period without undue depression of respiration, a period which should ordinarily be that of his deepest narcosis, surely uncontrollable depression should not occur several hours later. There must be some other factor which enters in; and while obstruction to respiration is not mentioned in these cases, it has always seemed to me that it must be this other factor. If this be assumed, the sequence of events becomes perfectly logical. During operation the patient is under the immediate observation of the anesthetist and his airway is kept clear. When he is returned to bed, although still deeply narcotized, he is not under quite such close or such competent observation. It is then perfectly possible and indeed probable for his tongue to fall back in his throat and cause slight obstruction to respiration. This need be only enough in combination with the narcosis to prevent the patient from quite satisfying his need for oxygen. The deficit need be but small, although in time the combined deficits mount, to cause general anoxemia. This starts a vicious cycle similar to that mentioned above, but with the sinister addition of obstruction.

It may be objected that obstruction would be observed and corrected. True, it would be if at all marked. A very slight degree, however, might easily escape detection. It is this sort of obstruc-

tion, so slight that it takes hours to do its work, to which I refer and which is really more dangerous than more marked obstruction, which would be appreciated and corrected.

This may all sound theoretical, but I know that it can occur because I have seen exactly such a case as described. In this case a slight grade of obstruction, acting over a period of some six or seven hours, finally caused anoxemia so severe that it would undoubtedly have proved fatal had it not been corrected; yet it entirely escaped the attention of two experienced and capable nurses and of a physician especially summoned because of the patient's critical condition.

Slight injury from this cause is probably quite common. If the patient is otherwise in a serious condition, however, it may be very important. Severe injury is less common, while death due solely and entirely to this anoxemia is probably quite unusual. All these harmful effects of anoxemia, however, may be easily and completely prevented.

It is fairly evident that there are some disadvantages to rectal anesthesia and that there are a number of ways in which it may go wrong. The chief difficulties are the lack of control which the anesthetist has over the course of events, the fact that susceptibility to shock is increased, and the ease with which anoxemia may occur and initiate a chain of disastrous results. It is therefore not an anesthesia which can be applied routinely or be administered and watched by anyone. Some patients should not receive it at all, namely those who are greatly enfeebled, whether from age or disease, those with marked incapacity of liver or kidneys (remembering that a too low carbohydrate diet produces incapacity of the liver), and those especially with any sort of breathing difficulties, particularly obstruction to respiration. It should not be used if the operation is of a nature which may easily lead to surgical shock. The dose should be carefully adjusted to the condition of the patient, always staying on the safe side, and, last but not least, the patient should have careful oversight during the course of anesthesia, with special care in the avoidance of all anoxemia. Care should certainly extend through the postoperative period until the patient is definitely regaining consciousness and has full control of his throat reflexes and muscles. Unless he is to be under close postoperative supervision of known competence, adequate airways should be left in place at the conclusion of the operation.

While, like many other drugs and methods in medicine, there are distinct possibilities of trouble in the rectal use of these drugs, if they are employed with reasonable care and discretion they are safe and of distinct advantage as an aid to other anesthetics. They smooth the course of anesthesia, they reduce the amounts of other toxic drugs, such as ether and cyclopropane, and they increase considerably the comfort of the patient both before and after operation. They are of particular advantage in the presence of an uncontrollable source of ignition such as diathermy or x-ray, since they allow the use of nitrous oxide without anoxemia and without the addition of combustible agents such as ether or cyclopropane. They should not be regarded as *anesthetic* agents capable of producing a complete anesthesia of their own, but rather as *narcotic* agents which act as strong aids to the real anesthetics which follow them.

Briefly then, these drugs have distinct possibilities of danger if applied incorrectly or carelessly, but if used with thorough appreciation of their shortcomings they are safe and form valuable adjuncts to other anesthetics.

DRUGS FOR INTRAVENOUS ANESTHESIA

The make-up of this group is very similar to that of the first group. The two drugs most commonly used are the sodium salt of n-methylcyclohexenylmethyl-barbituric acid (Evipal) and sodium ethyl-l-methylbutylthiobarbiturate (Pentothal). Paraldehyde is also used to a moderate extent, and Avertin and other drugs may be used. Since these drugs are administered by injection into a vein, absorption into the circulation is instantaneous and runs parallel with administration. Elimination proceeds as with rectal administration. Thus, the balance between absorption and elimination and hence the depth of anesthesia are determined almost entirely by the rate of administration. When administration is stopped, absorption ceases and the plane of anesthesia does not continue to deepen as with rectal administration. In fact, the plane immediately begins to lighten. If the operation is but momentary and administration is therefore continued for but a few minutes so that the patient's body does not become saturated with the drug, recovery is very rapid and the patient regains consciousness in but a few minutes. If the administration is prolonged as for an hour or two, recovery is much more prolonged. The patient may recover consciousness in approximately half an hour, but remains somnolent for two or three hours. Hence the duration of recovery and thus the degree of control over anesthesia approximate that obtained with ether.

Paraldehyde, as with its rectal administration, is eliminated partly through the lungs. The irritation so caused practically always produces a good deal of coughing during induction, accompanied by the formation of mucus. The barbiturates, Evipal and Pentothal, tend to contract the bronchi.

The effects of these drugs on circulation and respiration are similar to those produced by rectal administration except that they appear much more rapidly and may be more marked. It is exceedingly easy to stop respiration entirely, and this is very likely to happen unless some care is taken. Usually pulse volume is well maintained and blood pressure is not unduly depressed unless large doses are rapidly administered. If this is done, collapse may occur, and in extreme cases the patient may die almost at once. It is therefore extremely important to be cautious during induction and to proceed slowly until it is definitely determined how the patient will react to the administration. A dangerous dose of these drugs is dependent not so much on its total size as on the rate of injection.

Since we have a far greater degree of control over the depth of anesthesia, a control which closely approximates that with ether, there is not the same danger attached to a deep plane of anesthesia by intravenous injection that there is to one by rectal injection; for if it becomes evident at any time that anesthesia is too deep we know that we can immediately start lightening it simply by stopping the injection, whereas with rectal anesthesia we have no such ability. On the other hand, an outstanding danger of the intravenous method is the tremendous rapidity with which anesthesia may be induced or deepened. Probably by no other method can such rapidity be even approached. While this is a valuable asset for those who will use this power with due care, it is a grave danger if it is not appreciated or if it is used indiscreetly.

In applying this method of anesthesia the same general principles hold as with the rectal method. The method should not be applied to those who are enfeebled or those with marked liver or kidney disease, and least of all to those with dyspnea, respiratory obstruction or other breathing difficulties. Since the barbiturates tend to contract the bronchi, this method is unsuitable and possibly dangerous to use with asthmatics. Operations where shock is likely to ensue should be avoided, although the need is not so great with this method as with the rectal methods, because control of depth is so much better. It is unsuitable for bronchoscopy and to a less extent for laryngoscopy, because it tends to contract the bronchi and is somewhat uncertain in abolishing the gag reflex.

The depth of anesthesia should be adjusted carefully to the tolerance of the individual and the requirements of the operation. Most patients readily withstand marked depth if it is maintained for but a short period, and withstand a prolonged period if anesthesia is light, but it is doubtful whether it is advisable to employ a deep plane of anesthesia for any considerable time. As with the rectal methods, a free airway must always be carefully maintained and anoxemia must be studiously avoided. A gas machine or other means of applying oxygen for inflating the lungs should always be at hand.

There are a number of technical details attention to which will make all the difference between a smoothly running procedure and one which is quite disturbing or even dangerous. For instance, there is sometimes during induction severe spasm of the larynx, which may in some cases produce anoxemia of such severity as to throw the patient into a critical condition. This spasm appears to be due to the inhalation into the larynx of saliva or mucus from the throat. As the patient goes under the anesthesia relaxation of the muscles of the throat takes place with great rapidity, and often complete obstruction from this cause ensues. The patient tries to breathe several times against this obstruction. When it is finally relieved by the anesthetist the patient thereupon takes a deep breath and thus pulls into his larynx any mucus or saliva which is near. If before induction is started the pillow is taken out from under the patient's head and his head is extended so that any fluid in the throat will tend to gravitate into the nasopharynx, and if, in addition, a free airway is constantly maintained as he goes under, spasm of the larynx will rarely occur.

These remarks on correct usage are not in any way intended to be complete, but are intended simply to show how apparently minor technical details may influence profoundly the smoothness or even the safety of this anesthesia, and thus to show some of the reasons why the anesthetist employing it should have had good training and be thoroughly familiar with this method.

The most important reason for employing these drugs arises from the fact that they are entirely noninflammable and nonexplosive. To appreciate the importance of this it is necessary to realize that fires and explosions are frequently taking place, chiefly from the trusted and much used drug, ether. This drug is readily inflammable and is explosive in certain combinations, and is treacherous in use because of the cool ether flame, which travels slowly and usually imperceptibly until an explosive mixture is reached. Combinations with oxygen or nitrous oxide may be highly explosive. Nonvolatile and noninflammable drugs such as these, and their combination with nitrous oxide, form practically the only means of obtaining a noninflammable and nonexplosive general anesthesia without anoxemia, and hence, with the increasing use of high-frequency, high-tension electrical apparatus in the operating room, are of the

greatest importance and usefulness. They also contribute greatly to the comfort of the patient by lessening preoperative apprehension and often postoperative vomiting. The intravenous method produces a desirable anesthesia for short procedures where deep relaxation is needed, as in the manipulation of joints, because induction is easy and rapid, relaxation is excellent (with Pentothal), and recovery is rapid and usually unaccompanied by nausea or vomiting.

LONG-ACTING SPINAL ANESTHETICS

Within the last few years two new drugs, Pontocain and Nupercain, have come into use for spinal anesthesia, but have been met by some criticism. It is claimed that both these drugs are much more toxic than novocain and therefore should not be used. This criticism has been leveled particularly at Nupercain, which is said to have a dangerous action on the heart. Pontocain is said to have an especial affinity for the respiration and to produce respiratory paralysis.

It is undoubtedly true that these two drugs are many times more toxic than novocain, the toxicity of Nupercain exceeding even that of cocaine. Lundy in particular concludes from clinical observation that with Nupercain the margin between respiratory and cardiac failure is dangerously narrow.

Both drugs, however, are much more powerful than novocain, Nupercain being about ten times as potent as cocaine and twenty times as potent as novocain, and their dosage is correspondingly smaller. Thus when due allowance is made for their greater potency and corrected dosage is considered, they appear to be little more toxic than novocain. Moreover, the clinical toxicity of the local acting drugs is greatly reduced by dilution. Nupercain is most commonly used in 1:1500 dilution, and its clinical toxicity is thus greatly reduced.

Another aspect of toxicity is important in considering drugs for spinal anesthesia. Locally acting anesthetic drugs produce their toxic effects in two different ways: because of their presence in the general circulation and secondarily through paralysis of nerves. The first effect, toxic symptoms from absorption into the general circulation, is most prominent in local and regional anesthesia because the amount of drug used in these methods is comparatively large. Toxic symptoms from absorption are not so marked with spinal anesthesia, simply because the dose of the drug is usually comparatively small. For trans-sacral anesthesia, for instance, nearly 1 gm. of novocain would probably be necessary, but for the same anesthesia by the spinal method, 100 mg. or less of novocain, or approximately one tenth of the dose necessary in regional anesthesia, would probably be ample. Therefore, although absorption from the subarachnoid space is quite rapid, the amount which can be thus absorbed can hardly be as great as that by the regional method. Although absorption into the general circulation undoubtedly takes place in spinal anesthesia, and has its effect, the symptoms observed are probably due in much greater measure to the effect of nerve paralysis. Symptoms due to the presence of the drug in the general circulation are probably minor because of the small amount used.

It is thus apparent that in local and regional anesthesia the drug employed should be as nontoxic as possible, but that in spinal anesthesia this aspect is not so important as is the effect of the drug through nerve paralysis. Novocain is generally felt to be the least toxic of the locally acting drugs which we have at our disposal, and is therefore pre-eminently fitted for use in local and regional anesthesia. In clinical use in spinal anesthesia, however, it appears to have a more marked effect on the vasomotor system than do either Pontocain or Nupercain. At least that is the interpretation placed on the fact that in spinal anesthesia for a comparable extent and length of anesthesia it produces more depression than do either Pontocain or Nupercain.

In regard to the second criticism that Pontocain has a special affinity for the respiratory system, this belief arose during the early use of the drug, before its peculiarities in mixing and diffusion in the spinal fluid were understood. No one well experienced in its use now believes this. A method of use ill adapted to this particular drug caused it to run high in the spinal canal, but this ceased as soon as appropriate methods had been worked out.

Thus it appears that these drugs are not only as safe for spinal anesthesia as is novocain, but are actually safer, especially for comparable length and extent of anesthesia. Moreover, the much greater length of anesthesia attainable is of separate and distinct value in itself and may even on occasion be a factor of safety, especially in abdominal operations, for it adds distinctly to danger in these operations both from a surgical and from an anesthetic viewpoint to have spinal anesthesia run out in the middle of an operation and to have general anesthesia induced.

That these facts are appreciated by anesthetists is shown by the rapid rise to favor of these drugs. Nupercain in particular has gained world-wide repute, though Pontocain is perhaps used more frequently in this country. At the Lahey Clinic we do not now use novocain at all for spinal anesthesia, employing Pontocain for most cases and Nupercain for those in which operation is expected to last two or three hours.

SUMMARY AND CONCLUSIONS

It is fortunate indeed that ether was the first anesthetic discovered and put into general use, for while results were naturally improved by practice and experience, it could be given with a fair degree of safety by the inexperienced. Of the drugs which have been added since that time one might say that they have been increasingly specialized. They have certain distinct advantages over ether, often in a narrow field, but often they have certain special dangers also. Thus, they are not appropriate for routine use by everyone, but should be employed only by those who have had a good general training in anesthesia and who are thoroughly familiar with their special peculiarities and dangers.

The drugs under discussion belong to this group. Rectal anesthetics suffer from the almost complete lack of control over the course of events once the dose has been administered. The intravenous anesthetics harbor great possibilities of danger in the extreme rapidity with which anesthesia may be induced and deepened. With both of these, conditions are favorable to the development of anoxemia, sometimes of severe degree. With experience, care and attention, however, all these difficulties may be readily avoided. These drugs are of the utmost value in the presence of uncontrolled sources of ignition and are of some value in other respects.

Pontocain and Nupercain produce spinal anesthesia of much longer duration than does novocain and produce less vascular depression for a similar extent and duration of anesthesia. They should be employed only by those thoroughly familiar with their use, but are safe and of great value when so used.

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DISCUSSION

DR. SIDNEY C. WIGGIN, Boston: Dr. Sise has well stated the dangers in the use of the drugs which are placed in the rectum, veins and spinal canal, and his account leads us to conclude that their use should not be made routine but should be limited to selected cases. Rectal and intravenous drugs will eliminate the fears of the patient and the difficulties of induction. But, by the use of premedicating drugs to develop a basal anesthetic state, the same result is obtained and the attending dangers are less.

As Dr. Sise states, the rectal drugs allow for very little control of the depth of anesthesia and postoperatively leave the patient out of control of his respiratory reflexes too long, with the attending dangers of respiratory complications and anoxemia.

Avertin is most useful in little children under eight or ten years of age. This age group is more difficult to premedicate with the barbiturates and opiates than are adults. The little child benefits from rapid induction in his bed, recovers much more rapidly from the effects of Avertin and supplementary anesthetics and is not so prone to develop pulmonary complications as is the adult patient.

Rectal drugs are often chosen for the nervously unstable patient, in the hope that the trying experiences of induction and postoperative nausea and vomiting will be eliminated. In my experience the incidence of nausea and vomiting in this type of patient is greater with rectal basal anesthesia than with nitrous-oxide, oxygen and ether mixtures. Spinal anesthesia almost totally eliminates this complication.

An early indication for rectal drugs was the prolonged orthopedic operation. If spinal fusions requiring four or five hours can be better done under nitrous-oxide, oxygen and ether or cyclopropane and oxygen anesthesia, with the patient conscious soon after the operation, why use a more uncontrollable technic? In operations where an excessive loss of blood is anticipated, as in trachelorrhaphy or those for perineal repair or cancer of the rectum, the use of rectal or intravenous drugs will create an extreme state of circulatory depression.

The intravenous drugs, Pentothal and Evipal, are indicated in selected cases principally for emergency and short operations, administered fractionally. The safest and most controllable method of using them is in basal dosages, supplemented by nitrous-oxide and oxygen.

I agree with Dr. Sise that novocain used for spinal anesthesia in the dosages and concentrations required for maximum height, depth and duration shows more toxic signs than Pontocain or Nupercain. The Department of Anesthesia at the Lahey Clinic has shown good results with Pontocain and Nupercain. At the Faulkner Hospital our results with novocain used in minimum doses with varying doses of Pontocain for height and duration have been most satisfactory.

In using any spinal anesthetic for operations of long duration and for shorter operations in the upper abdomen, one should recognize three types of patients: the good-risk patient who can stand prolonged spinal anesthesia; the borderline-risk patient with debility and varying sorts of complicating diseases, who should not be subjected to the dosage required for a deep, high spinal anesthesia; the bad risk patient, with whom we are all familiar, who should not receive spinal anesthesia under any consideration. The borderline-risk patient should receive partial spinal anesthesia with a very moderate dosage of the agent, and, using this as a basal anesthesia, should be given supplementary anesthesia with a mixture of cyclopropane and oxygen. This combination gives complete relaxation, with the patient under control of a high oxygen concentration. The bad-risk patient should receive nitrous-oxide and oxygen and local infiltrations with novocain.

In conclusion I should like to emphasize some of the points to which Dr. Sise has referred. First, before using these agents, the physician should recognize the contraindications to spinal anesthesia, which are as follows: marked, debilitated states from acute and chronic disease, sepsis, shock and hemorrhage; hypertension with cardiac and renal disease; and severe cardiac conditions. Intravenous agents have in addition the following contraindications: toxic thyroid disease; and bilateral kidney disease. Rectal drugs have the same contraindications as those used for intravenous anesthesia, as well as being prohibited by certain diseases of the rectum. Secondly, the patient should be evaluated for the choice of premedication drugs, anesthetics and method of administration from the history, physical examination, laboratory findings and type of operation. Thirdly, in administering the agents, care should be observed in keeping them at a minimum dosage for the patient and the operation and in maintaining complete oxygenation of the patient throughout. These drugs should be administered only by those well acquainted with them.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24361

PRESENTATION OF CASE

A sixty-nine-year-old white, American confectioner entered the hospital with the complaint of pain in his legs of two months' duration.

The patient was in a semicomatose condition so the history was obtained from his wife. Two months before entry he noticed the gradual onset of sharp pain running down the back of his right leg to the knee, and one week later a similar but less severe pain in his left leg. The pain was aggravated by walking and moving his legs, and it could not be relieved. His physician made a diagnosis of sacroiliac disease and prescribed codeine and massage. One month before entry he went to an outside hospital where x-rays were said to show arthritis of the spine. A plaster cast was applied over the lower part of his body which caused sharp severe pain at the tip of the right scapula when he lay down. As a result he slept in a Morris chair. Soon after the application of the cast he developed marked edema of his legs up to the hips, with pain and tenderness in his right leg. Two and a half weeks before entry he began to have periods when he perspired profusely, although there was no change in the character of his pain or other symptoms. A few days later he was fitted for a brace to take the place of the cast. This caused him no particular discomfort, but twelve days before entry his left leg became intensely tender to touch and he had severe pain in the upper part of it when he walked or moved it. He remained in bed from then until he entered this hospital. Nine days before entry he went back to the outside hospital by ambulance to get his brace and was advised to enter the hospital for treatment of heart disease, but this he refused to do. At that time he only had pain in his left leg. One week before entry he became incontinent of urine, and the following day he had some dyspnea and a substernal choking sensation. Two days later he became irrational and delirious and began to show twitching of his muscles.

Previous to his present illness he had had no dyspnea, cough, edema or neuromuscular or genitourinary symptoms. At the age of five he had had "rheumatism" which was said not to have been rheumatic fever. For many years he had had indigestion and chronic constipation, but no abdominal pain or unusual appearance of his stools. For about five years he had complained of "nervous exhaustion" which was relieved by taking "neurophosphates." He had not lost any appreciable amount of weight.

Physical examination revealed a well-developed and nourished man who was somewhat disoriented and drowsy. The heart borders were not made out, and the sounds were distant. A systolic murmur was heard at the left of the sternum. The rhythm was regular. The blood pressure was 128 systolic, 64 diastolic. The lungs were negative throughout except for some emphysema and a few fine rales at the left base. The abdomen was negative. There was marked atrophy of both legs and pitting edema of the left foot. There was also slight edema of the sacrum with a decubitus ulcer on the right buttock. A neurological examination revealed generalized twitching of the muscles of the face, lips, tongue, arms and legs. There was marked arteriovenous nicking of the vessels of the fundi, but no exudates or hemorrhages. Examination of the cranial nerves and of sensory sensation all over the body was unsatisfactory because of the muscle twitching and mental disorientation. No reflexes of any kind could be obtained.

The temperature was 101°F., the pulse 105. The respirations were 23.

The urine showed a maximum specific gravity of 1.012 and contained a slightest possible trace of albumin with 2 to 10 white cells and 1 to 5 red cells per high-power field in the sediment. It contained no Bence-Jones protein. The blood showed a red-cell count of 4,100,000 with 70 per cent hemoglobin and a white-cell count of 18,300 with 82 per cent polymorphonuclears. A few nucleated red cells were seen in the smear, but no plasma cells or primitive white cells were observed. The guaiac test on the stool was 2+. The nonprotein nitrogen of the blood was 50 mg. and the protein 5.6 gm. per cent, and the blood Hinton was negative. An electrocardiogram showed inverted T_2 and T_3 and diphasic T_4, which may have been due to the digitalis which he was taking. The QRS complexes in Leads 1, 2 and 3 were slurred, and there was slight right-axis deviation. A lumbar puncture was done in the fourth lumbar space, and about 0.75 cc. of xanthochromic fluid was obtained with a syringe. The fluid clotted quickly. The supernatant fluid contained 30 lymphocytes per cubic millimeter. It gave a 4+ alcohol test for protein.

X-ray films of the spine and skull were of poor quality because of lack of co-operation on the part

of the patient. However, the seventh dorsal vertebra was markedly decreased in height and slightly widened. There were hypertrophic changes about several vertebrae, particularly marked in the upper lumbar spine. The skull was negative.

He continued to be in a semicomatose condition throughout his stay in the hospital. His temperature was maintained at the level of 101°F. for the first six days but rose gradually to 103°F. before his death. On the sixth day the nonprotein nitrogen of the blood was 77 mg. and the serum protein 3.9 gm. per cent. A urine culture showed an abundant growth of *Bacillus coli*. He was given 2 cc. of Salyrgan which failed to produce a diuresis. On the eighth day the nonprotein nitrogen had risen to 165 mg. per cent, and the chlorides were equivalent to 110 cc. of N/10 sodium chloride. He became more dyspneic and cyanotic, and his blood pressure fell to 80 systolic, 40 diastolic. The pulmonary congestion increased, but no signs of consolidation could be made out before his death on the ninth day.

Differential Diagnosis

Dr. Bernard M. Jacobson: If we had seen the patient early in his illness we might have suspected from the bilateral distribution of his pain that he had something the matter with him besides sacroiliac disease.

Apparently from what is stated a little farther on, the edema was considered to be related to heart failure. We have no other reason for believing that it was heart failure; at least no other reasons are given. I have no explanation whatever, either now or later, for this edema, because it was a symptom that apparently subsided eight days later.

There is no statement relative to the odor of the breath, something that we certainly should be interested in. In view of the fever that was found we can hazard a guess that these rales represented early bronchopneumonia rather than left-heart failure.

There are two important physical findings we should like to know about, and although they are not stated, I shall assume that they were negative. In the first place we are not told anything about palpable lymph nodes, and secondly, nothing about the feel of the prostate on rectal examination.

A lumbar puncture was done, and apparently the pressure was extremely low. The findings are those of complete subarachnoid block above the fourth lumbar space.

In the x-ray report we are not told anything about the amount of rarefaction in the vertebrae, if such were present.

X-Ray Interpretation

Dr. Aubrey O. Hampton: This is the deformed vertebra that was spoken of. That deformity is just about all it shows. I cannot see any bone destruction or soft-tissue mass. The margins of the vertebrae are smooth and sharp, and there is the general curve of an old, long-standing kyphosis.

Dr. Jacobson: Could it have been a compression fracture?

Dr. Hampton: Yes; and if so, might have nothing to do with the present disease.

Dr. Jacobson: Is it not true, that such a lesion can be present without any disability?

Dr. Hampton: Yes; and also without any knowledge of injury.

Dr. Jacobson: You cannot say there is rarefaction?

Dr. Hampton: No.

Dr. Jacobson: Is that hypertrophic arthritis?

Dr. Hampton: Yes; extensive hypertrophic changes around all the margins of the vertebrae. The skull appears to be normal.

Differential Diagnosis (continued)

Dr. Jacobson: On the basis of the history and physical examination one can make two clinical diagnoses. In the first place, bilateral pain in the legs, urinary incontinence and marked atrophy of the lower extremities at the time of admission certainly point to a low cord lesion or to one in the cauda equina. This is confirmed by evidence of complete block at the time of tapping. The lesion is probably at the level of the dorsolumbar spine. In the second place, the delirium, the coma and generalized muscular twitchings certainly indicate clinical uremia, and the blood chemistry with a rapidly advancing nonprotein nitrogen certainly confirms the clinical diagnosis of uremia.

Our next problem is to try to decide the nature of the spinal-cord and renal lesions. Disease of the spine itself, in a man of sixty-nine, is commoner than primary disease of the cauda equina. If this is spinal disease and if the seventh dorsal vertebra represents the lesion this process is probably a neoplasm with multiple deposits in the spine, despite the negative x-ray. In this location the commonest neoplasm in a man of sixty-nine is metastatic carcinoma of the prostate or of the gastrointestinal tract, but such a diagnosis does not seem likely. We assume that the prostate was negative. While it is true that one stool showed a 2+ guaiac test, this may well have been due to uremia and is not particularly suggestive of cancer of the gastrointestinal tract. Finally, I think it is rather rare for metastatic carcinoma in the spine

to be large enough to compress the cord without being visible by x-ray. Is that a fair statement?

DR. HAMPTON: We have seen several cases where the disease was in the lamina and involved the cord with no changes in the vertebral body; because of the difficulty in examination of laminae the x-rays have been considered normal.

DR. JACOBSON: With cord compression?

DR. HAMPTON: Yes.

DR. JACOBSON: Probably the vast majority do show x-ray changes.

DR. HAMPTON: I think so.

DR. JACOBSON: Probably the second commonest tumor of the spine in a man of this age is a myeloma, and x-ray certainly does not rule out this possibility. We are told that Bence-Jones protein was not found in the urine and that plasma cells were not seen in the blood smear. But those are two notes I do not believe we can accept with certainty. It is true that the serum protein is not elevated, but such a finding does not rule out myeloma because only about half the cases show hyperproteinemia.

I should like to turn to the nature of the renal lesion. It is a very unusual situation. In spite of the uremia the blood pressure was not elevated, and no evidence of congestive heart failure is found in the history. Chronic hemorrhagic Bright's disease and chronic vascular nephritis are unlikely, and the relatively sparse urinary sediment, in spite of the *B. coli* culture, is against a severe pyelonephritis. Is the renal failure due to passive congestion? Apparently that thought was entertained because he was given Salyrgan, but I do not believe we have other signs of cardiac failure, and the degree of nitrogen retention is far too excessive for renal failure to be due to passive congestion.

I should like to make my diagnosis cover both the renal and the spinal lesions. I know of only one disease that will involve the spine, cause compression of the cord and also involve the kidney, and that is multiple myeloma. The kidney lesions usually result in a terminal uremia, accompanied by normal blood pressure and a relatively insignificant urinary sediment. Of course there are a large number of objections, but I do not believe we want to take time to go into them. My diagnosis is multiple myeloma, with compression of the cord, Bence-Jones kidneys, congestive heart failure and terminal bronchopneumonia.

CLINICAL DIAGNOSES

Arteriosclerotic heart disease.
Myocardial insufficiency.
Spinal cord tumor, ? fracture of seventh dorsal vertebra.

DR. JACOBSON'S DIAGNOSES

Multiple myeloma.
Compression of spinal cord.
Bence-Jones kidneys.
Congestive heart failure.
Terminal bronchopneumonia.

ANATOMICAL DIAGNOSES

Carcinoma of the stomach with metastases to the liver, adrenals, kidneys, spinal cord, vertebrae and mediastinal, mesenteric and retroperitoneal lymph nodes.
Cardiac hypertrophy, slight.
Hydrothorax.
Bronchopneumonia.

PATHOLOGICAL DISCUSSION

DR. TRACY B. MALLORY: I think we are under suspicion of having set a trap for Dr. Jacobson because the combination of a spinal lesion and uremia certainly does suggest myeloma. The diagnosis in this case is not foreshadowed by anything that appears in the clinical history, the laboratory findings or the x-rays. He had a large carcinoma of the stomach — the primary source of all his difficulties — and multiple metastases from that. Nearly every vertebra was involved. The majority of them were almost cheesy in character, with hardly any bone at all left in the seventh dorsal. One or two of the lumbar vertebrae were extensively involved. The tumor had penetrated the posterior wall of the vertebral body and invaded the spinal canal quite extensively in the region of the seventh dorsal and to a less extent in the region from the twelfth thoracic to the second lumbar vertebra. I think there was enough intraspinal tumor there to explain the symptoms that suggested caudal involvement. The terminal event was a very extensive terminal bronchopneumonia.

A PHYSICIAN: What about the uremia?

DR. MALLORY: His heart weighed 425 gm., which is definite hypertrophy. The kidneys were moderately congested and showed minimal arteriolar changes, otherwise nothing.

DR. HAMPTON: I am not trying to defend myself or the x-ray department, but I want you to know that the diagnosis of spinal metastases from a lesion in the gastrointestinal tract is rarely made by x-ray. You can scrape out the entire inside of a vertebra, and so long as you leave the cortex, you cannot see anything wrong with it by x-ray. It has been done experimentally and is surprisingly true. The fracture I suppose was due to cancer, but it certainly does not look like it.

A PHYSICIAN: Did he have any phlebitis?

DR. MALLORY: No. The leg veins were exam-

ined and were entirely normal. The edema was not due to thrombosis of the leg veins.

A PHYSICIAN: Was there pressure on the renal veins by enlarged lymph nodes?

DR. MALLORY: There was a node in the neighborhood of one of the adrenals which conceivably could have pressed on the renal veins, but there is no particular reason to suppose it did. The adrenals were extensively replaced by tumor, as is often the case in stomach cancers; but there was still a fair amount of adrenal tissue left, so I do not believe that any element of Addison's disease entered the picture.

CASE 24362

PRESENTATION OF CASE

A fifty-one-year-old Portuguese housewife entered the Emergency Ward complaining of severe shortness of breath.

Thirty-three years before entry, when eighteen years of age, she had had rheumatic heart disease with fever, which lasted for four months. Except for slight shortness of breath on exertion she was essentially symptom free until four years before entry, when the dyspnea on exertion increased and was accompanied by a feeling of fullness in the neck. A year later she began having some precordial pain on exertion, which was relieved by rest. She was given digitalis at that time, which she continued to take at irregular intervals. Two years before entry she noticed a tendency to wheeze after eating or on lying down, and a year later began having definite attacks of paroxysmal nocturnal dyspnea. Two months before entry she had several attacks of severe stabbing pain in the precordium that came on during exertion, did not radiate and were relieved by rest. Shortly after that she omitted digitalis entirely. About a month before entry she noticed slight swelling of her ankles. This increased fairly rapidly until they were grossly swollen. A few days before entry she began to have pain in the back of the neck and in her left arm. She had no abdominal pain, distention or other symptoms. At the time of entry her dyspnea on exertion was so severe that she was unable to do any kind of work.

She had had severe yellow fever when in Brazil thirty years before entry and malaria when in New Orleans four years later, which lasted for one year. She had had frequent sore throats and said that she had had mild pneumonia on six different occasions. Fifteen years before entry she had been treated for pyelitis at an outside hospital. About three or four months before entry she had experienced an attack of amnesia lasting about three-quarters of an hour, during which she forgot her name, telephone number and the purpose of the errand which she was doing at the time. Her appetite had been poor for about a year before entry because eating caused abdominal discomfort. Her diet consisted of cereals, milk, canned fruits and salad, with very little meat. Her past history and family history were otherwise essentially negative.

The physical examination revealed a well-developed, somewhat undernourished, slightly cyanotic, chronically ill woman, propped up in bed breathing rapidly but in no acute distress. The neck veins were distended, and there was slight pitting edema of the ankles. There were a few moist rales at both lung bases. The heart was enlarged to the left, 11 cm. from the midsternal line in the sixth interspace. The sounds were of poor quality, and there was a gallop rhythm. At the apex there was a palpable systolic thrill, a short high-pitched systolic murmur and a diastolic rumble with presystolic accentuation. No aortic murmurs were heard. P_2 was louder than A_2. The pulse was regular except for frequent extrasystoles, and the blood pressure was 120 systolic, 80 diastolic. The liver was palpable two fingerbreadths below the costal margin.

The temperature was 99°F., the pulse 97. The respirations were 28.

The urine examination was negative. The blood showed a red-cell count of 4,660,000 with 90 per cent hemoglobin and a white-cell count of 13,500 with 65 per cent polymorphonuclears. Stool examination was negative. The nonprotein nitrogen of the blood was 25 mg. and the protein 6.1 gm. per cent. A blood Hinton test was negative. A blood culture was negative. An electrocardiogram showed flat T_1, diphasic T_2, T_3 and T_4, low T_4 and slight right-axis deviation.

An x-ray film of the chest showed the heart to be dilated on both sides, with a prominent pulmonary conus. The left auricle reached the right border of the heart. The hilus shadows were decreased in size, and the lung markings were slightly prominent. There was a localized area of dullness close to the lower border of the right heart. The costophrenic angles were clear, and the diaphragm was in normal position.

She was given Salyrgan and Mercupurin and proved to be sensitive to both. She was digitalized but failed to show much improvement. Soon after entry she began to raise small amounts of bloody sputum. On the fourteenth hospital day she began to have severe pleuritic pain in the right lower chest posteriorly, and her hemoptysis increased. An area of dullness with bronchial breathing could be made out at the right base, and a portable x-ray film showed the area of consolidation in the right cardiophrenic angle to be slightly

increased in size. The white-cell count rose to 40,000, and the temperature to 102°F. A blood culture taken on the seventeenth day contained *Staphylococcus aureus* in one of two flasks. She gradually failed and died on the twenty-second day.

Differential Diagnosis

Dr. Milton H. Clifford: We first note that the patient had rheumatic fever when she was eighteen years old, and that she had had no symptoms directly related to the cardiovascular system, except for slight shortness of breath on exertion, until the last four years before entry. This would indicate that, if she had had any heart damage at the time of the rheumatic fever, she had tolerated that damage extremely well from a functional point of view. The slight shortness of breath during the earlier years could well be consistent with a well-compensated mitral stenosis. Also, during the intervening years she had gone through several severe illnesses, and so far as one can tell from the records, her cardiac reserve was sufficient to have been unaffected by them, unless the alleged attacks of "mild pneumonia" on six different occasions were mis-diagnoses, that is, wrongly deduced from slight signs of decompensation at the time of severe colds. If this were the case, it is surprising that she continued to have as good cardiac reserve as she had. Significant progressive breakdown of cardiac reserve, then, seems to date from four years before entry.

Patients with mitral stenosis may have some precordial pain on exertion, and with the progressive pulmonary back pressure of the stenosis, vital capacity of the lungs decreases, accompanied with increasing dyspnea even up to the point of attacks of cardiac asthma. In view of this marked pulmonary hypertension, one might also expect that during the four-year period hemoptysis would occur on this basis. This, however, was not true with this patient. One also wonders about the possibility of aortic regurgitation, since one would expect paroxysmal nocturnal dyspnea more frequently with this valve lesion, or of myocardial weakness. She apparently did not get enough digitalis to judge any therapeutic effects, even if they could be obtained. Her attack of amnesia about four months before entry suggests that she may have had a cerebral thrombosis or, more likely, in view of her heart disease and the pulmonary infarction that we later find evidence of, an embolism that pushed on into the smaller radicles leaving no apparent residual. The anorexia and discomfort on heavy eating could not be considered unusual in any case of poorly compensated chronic heart disease, and I think it is quite unnecessary to look further for a cause of these symptoms.

The physical examination confirms the history of a decompensated heart, both right and left sided. The examination of the heart is consistent with mitral stenosis, but there is no indication of a lesion at the aortic valve. One wonders if the blood pressure was not previously higher, being at an apparently normal figure when the patient was in decompensation. The frequent extrasystoles, an apparently correct observation, confirmed at least by no mention of fibrillation in the electrocardiographic report, could be present in rheumatic heart disease, though they are not typical of decompensated mitral stenosis. Her practically normal temperature and negative blood cultures lead one away from thoughts of a complicating subacute bacterial endocarditis.

The laboratory work is of interest only from the fact that the patient did not show the relative polycythemia which might be expected with the prolonged venous back pressure.

The x-ray film of the chest is perfectly consistent with mitral stenosis. One is surprised, however, not to find greater signs of pulmonary congestion or even some fluid. The localized area of dullness close to the lower border of the right heart is hard to explain, save on the possibility of an infarct, especially in view of the later reports. If the hemoptysis that began shortly after entry was due to the pulmonary congestion from mitral stenosis alone, one might have expected this x-ray to show more evidence of pulmonary congestion than it did.

The electrocardiogram showed right-axis deviation and T-wave changes. So far as the right-axis deviation is concerned, it is consistent with the diagnosis of mitral stenosis. It is not so easy to interpret the T waves. They are certainly abnormal and indicate either digitalis change, — although so far as was known she had not had digitalis recently, — myocardial degeneration or actual coronary disease.

If a patient has gone for over thirty years with good cardiac reserve in a heart that was presumably injured during an attack of rheumatic fever, one can readily presuppose that damage from degenerative changes and premature arteriolarsclerosis can play a greater part in bringing about late decompensation than the damages of the rheumatic heart disease per se. Also, the poor results of digitalization on what is presumably the first decompensation suggest a very poor myocardium. The anginal attacks the patient had during the last few years before entry, and particularly the attack she had two months before entry, although consistent up to a moderate degree of

severity with mitral stenosis alone, suggest also the possibility of coronary heart disease. Again, one is surprised at the apparently normal blood pressure and wonders if she has not run a definitely higher one before her acute illness brought her to the hospital. And again, the electrocardiogram could be considered consistent with coronary heart disease and myocardial damage. The source of the apparent pulmonary emboli and the probable cerebral embolus four months earlier can only be presumed. In view of the fact that she had no evidence at any time of fibrillation, one is led away from the probability of thrombi in the auricles. She could, however, have a mural thrombus from an old infarction at the apex of the heart.

Clinical Diagnoses

Rheumatic heart disease.
Multiple pulmonary infarcts.
Bronchopneumonia.

Dr. Clifford's Diagnoses

Coronary heart disease with infarction.
Pulmonary infarcts.
Rheumatic heart disease with mitral stenosis.

Anatomical Diagnoses

Rheumatic heart disease with mitral stenosis.
Ball thrombus, left auricle.
Pulmonary infarction, multiple, massive.
Pleuritis, acute fibrinous, bilateral.
Arteriosclerosis, minimal, aortic and coronary.
Coarctation of aorta, minimal.
Infarcts of kidneys.
Thrombosis of perivesical, perivaginal and inferior hemorrhoidal veins.
Thrombosis of right popliteal and right posterior tibial veins.

Pathological Discussion

Dr. Tracy B. Mallory: The postmortem examination on this patient showed an almost pure mitral stenosis. The valve was reduced to a very narrow slit, 1.5 cm. in length and barely wide enough to admit the insertion of a thin knife blade. The heart weighed 325 gm., which for a small woman represents a slight but definite degree of hypertrophy. It was, however, quite markedly dilated so far as the two auricles and the right ventricle were concerned. The left ventricle appeared slightly atrophic. There was no significant degree of coronary sclerosis, and the myocardium was free from scars or areas of infarction. All the other valves were entirely negative. There was one quite unusual cardiac finding, however. Lying entirely loose in the left auricle was a spherical ball-like thrombus with a rough, grayish-white surface and marked lamination on cross section. It must have been present in the auricle for a considerable period of time. Presumably it originally arose as a mural thrombus, very likely in the auricular appendage, but all trace of such connection had been lost. What part it may have played in the symptomatology I do not know. It was too large to have slipped into the mouth of the mitral valve and to have acted as a ball thrombus, but one can readily imagine its being sucked down over the mouth of the valve, which it would have covered completely in such a manner as to have completely obstructed the circulation. Fragments from it presumably accounted for the cerebral embolus which Dr. Clifford suspected and for the two emboli that produced infarcts in the kidneys.

The most extensive pathologic changes, and the ones which I feel unquestionably were most responsible for her rapid downhill course, were in the lungs. We counted fourteen separate infarcts scattered fairly uniformly throughout all five lobes. Many of them were quite large, ranging from 5 to 9 cm. in diameter. The total area of infarction must have added up to about half of the entire lung volume. Only one of the infarcts was very fresh; the majority seemed to have been present for some period of time, certainly two weeks and possibly as long as four weeks. The emboli responsible for the infarcts were widely distributed throughout the small and intermediate-sized pulmonary arteries. The major pulmonary branches were free. Two possible sources for the pulmonary emboli were found. There was extensive thrombosis of veins throughout the pelvis, particularly the perirectal and perivaginal plexuses. Thrombi were also found in the right popliteal and posterior tibial veins.

One other anatomical finding was of interest because of its rarity. There was a slight degree of coarctation of the aorta. It was too slight, I believe, to have played any role in the symptomatology, particularly since the left ventricle was not hypertrophied and no evidence of dilated collateral circulation could be made out. Finally, I doubt if the patient ever had hypertension of a significant grade, because the kidneys, except for the infarcts, were quite normal and there was no hypertrophy of the left ventricle.

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A FOUR-POINT PROGRAM

EXISTING surveys in regard to the adequacy of medical care and the personal experience of many practitioners have established without doubt that in some communities the medical care for the indigent is inadequate. It is also a fact that the proper cost of good medical care is too high to permit individuals in the low-income group to remain private patients unless some provision can be made to pay for such care.

These two factors have aroused a considerable sentiment throughout the country to have the Government add to its already existing medical activities the supervision of the practice of medicine. The great majority of the medical profession feel that this would be a mistake, but unless some constructive program can be put into operation to furnish adequate medical care for those of the indigent who are in need of it and to provide a means for the individual in the low-income group to pay the costs of illness, these calls for governmental interference will continue. If the following four-point program were developed in each community, the care of the indigent and financial burden of illness would be provided for and each community would then be able to determine whether it needs outside financial aid for the care of its indigent and low-income groups. This program will naturally vary with the size and make-up of the community, the density of the population, the means of transportation, and so forth. Each community must have: a well-equipped and properly staffed hospital for all strata of society; an insurance service to cover hospital charges; an insurance service to cover physicians' fees; and a health-service council composed of the physicians and all other groups interested in local health problems.

Already, most communities, particularly in New England, are well supplied with hospitals, but too often they are poorly equipped and staffed. Equipment should be modernized, and the hospital should be open to all properly trained, reputable physicians in the community; but the activities of the latter should be closely supervised, as in the case of the "teaching" hospitals with limited staffs.

Already, there are several insurance plans to cover hospital charges, but unfortunately these are only available to certain groups of the employed. The service must be extended to include all in need of it.

Already, there are insurance schemes to pay physicians' fees, but there seems to be a tendency on the part of organized medicine to combat some of them. No one plan will be ideal for all communities, and organized medicine should therefore encourage the development of a variety of schemes in order that the most satisfactory one will eventually be found.

Already, the establishment of health-service councils has been urged by the Council of the Massachusetts Medical Society, but the district societies have shown little inclination toward taking this

step which is so vital to the determination of the medical needs in a given community.

The above four-point program, or some modification thereof, should be adopted so that the people in the indigent and low-income groups in Massachusetts will not add their voices to those of many throughout the country who are asking for the Government to take over the practice of medicine.

EYES ON THE FUTURE

THE twenty-third annual report of the National Society for the Prevention of Blindness, Incorporated, under the above title, records both the passing of three decades since the sight-saving movement was organized in 1908 and the centenary of the birth of Louisa Lee Schuyler, the founder of the society.

Miss Schuyler, horror-stricken when she learned that thousands of babies became needlessly blind because of the omission of prophylactic treatment at the time of birth, was one of ten who established a New York State Committee on Prevention of Blindness in 1908. These pioneers were mainly interested in fighting ophthalmia neonatorum, then a major cause of blindness, but from this beginning grew the national society, with its 18,000 members and donors and its services extending from the protection of the eyes of the newborn to the preservation of sight in old age.

During the year 1937 the society supported in a number of ways the nationwide effort, led jointly by the United States Public Health Service and the American Social Hygiene Society, to make syphilis the "next great plague to go." It records a new high in the number of students enrolled in the summer courses for sight-saving teachers, one hundred and twenty having attended the courses given at various universities. Forty-four new sight-saving classes were established, so that by the end of the year five hundred and fifty-eight were in operation in the United States and Hawaii.

Other activities have included participation in the first session on eye health ever included in the annual meeting of the American Student Health Association; continued work on a revision of the standards of lighting for schoolrooms; an annual campaign for the "safe and sane" celebration of the Fourth of July; and further work on the revision of the classification of the causes of blindness by the Committee on Statistics of the Blind, sponsored jointly by the society and the American Foundation for the Blind. Educational work has been furthered by exhibits and displays, by the distribution of publications, by the showing of films and slides, and by the sponsoring of newspaper and magazine publicity.

The Society for the Prevention of Blindness is one of a number of organizations on which our country can look with pride. Conducted and supported by inspired and devoted followers, it works toward a worthy goal with complete unselfishness.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

RUPTURE OF THE UTERUS AT TERM

Mrs. A. B., a thirty-eight-year-old gravida V, at term, was seen in consultation on June 15, 1922, when she was in labor.

The family history was essentially negative, as was the patient's past history. She had had no operations and no serious illnesses. Catamenia began at twelve and were regular, with a twenty-eight-day cycle, lasted four to five days and were unaccompanied by pain. Her last period began on September 8, making her expected date of confinement June 15. Her previous pregnancies and labors had been normal, and she had had no intrauterine manipulation.

A physical examination at the beginning of her pregnancy was perfectly normal, and her prenatal course was uneventful.

She started in labor about 6 a. m. and came directly to the hospital. Labor progressed normally, and a vaginal examination at 11 a. m. showed the cervix to be almost fully dilated. The membranes were ruptured artificially, and the head was said to be in the pelvis. Shortly after

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

noon the pains became irregular, and about 2 p. m., while walking from her bed to a chair, she experienced an excruciating pain in the abdomen and fainted.

When seen at 3:45 p. m., the patient was in profound collapse. She was restless, pale and perspiring. Her pulse was thready, with a rate of 140. The blood pressure was 90 systolic, 70 diastolic. Palpation of the abdomen revealed a hard mass in the left lower quadrant apparently continuous with a softer one on the right, which rose almost to the ribs; there was a definite sulcus between these two masses in the median line below the umbilicus. No fetal heart was obtained. There was a small amount of fresh vaginal bleeding. A rectal examination revealed no presenting part. The diagnosis lay between a spontaneous rupture of the uterus with the baby lying free in the abdominal cavity or a twisted ovarian cyst. Laparotomy was decided upon, and as soon as the patient had been transfused, her physician acting as donor, the operation was performed. A low median incision was made with the escape of a moderate amount of free fluid blood. The baby and placenta were found in the abdominal cavity. The hard mass on the left proved to be the uterus, firmly contracted and inside out, which had ruptured posteriorly from the top of the fundus to the lower segment, the rent extending through the posterior cul-de-sac into the vagina. Hysterectomy was deemed to be the safest procedure and was performed. A gauze drain was placed through the posterior cul-de-sac into the vagina.

The convalescence for the first ten days was very stormy. The patient developed sepsis and a low-grade peritonitis which cleared up without the formation of pus that required surgical treatment. She was discharged on the fortieth day.

Comment. Any patient who, while in labor, suffers an acute pain that results in collapse and in whom the presenting part no longer remains in the pelvis must have a ruptured uterus. No other condition will allow the displacement of the presenting part.

The past history of this patient revealed no possible etiology for the rupture. She had had no intrauterine manipulation; there had been no miscarriages; there had been no curettages; there had been no adherent placentas; and the menstrual history had always been perfectly normal. It is possible that a Bandl's ring existed at the time the membranes were ruptured. This, however, is problematical.

Laparotomy is the operation of choice as soon as transfusion can be performed and a diagnosis of ruptured uterus made. Sometimes, as in the rupture of a previous cesarean scar, the rent in the uterus may be sewed as one sews up an initial cesarean. In this case it was impossible to suture the uterus as contractions had practically turned it inside out. It is interesting to note that at the time of operation there was practically no bleeding. Hysterectomy was the only operation that met the requirements in this particular case. The drain was undoubtedly inserted because of the rent in the vagina; otherwise, drainage is not only unnecessary but probably harmful.

DEATH

SNOW — Asa V. Snow, M.D., of Cushman, Massachusetts, died June 8. He was in his eighty-first year.

Dr. Snow received his degree from the Bellevue Hospital Medical College in 1879. He was a former member of the Massachusetts Medical Society.

MISCELLANY

USE OF IMPINGER METHOD FOR DETERMINATION OF AIR DUSTINESS

The emphasis recently placed on the problem of harmful dusts encountered in various industries has caused a marked increase in the number of persons interested in the determination of air dustiness. The many requests for information on the determination of the concentration of dust in the air indicated the need for a detailed description of the impinger method as used by the Bureau of Mines, United States Department of the Interior. A report just issued by the bureau contains a detailed description of the impinger method, with a history of its development.

The impinger was developed by the Bureau of Mines, in co-operation with the United States Public Health Service, during the course of an investigation of dust-sampling instruments in common use.

The method has been used widely for collecting data on air dustiness in mines and industrial establishments in the United States. Owing to the fact that permissible limits have been established on the basis of impinger results, the impinger method has been rather widely accepted as the standard in this country.

Possible sources of error and limitations of the impinger method are given in the report, so the true significance of the impinger results will be understood, thereby aiding in the proper interpretation of the results.

An appendix is included, which lists the publications dealing with results obtained by the impinger method, equipment needed for using the impinger method, and information that should be collected during sampling.

Copies of this report, Information Circular 7026, "A Technique for Use of the Impinger Method," by Carlton E. Brown and H. H. Schrenck, may be obtained from the Bureau of Mines, Washington, District of Columbia. — *U. S. Department of the Interior.*

SQUIBB INSTITUTE FOR MEDICAL RESEARCH

Organization of the Squibb Institute for Medical Research, in which a staff of scientists assembled from leading institutions of the United States and foreign countries will attack problems involved in the cure of disease and relief of pain, is announced by E. R. Squibb and Sons.

The institute will be housed in a new laboratory building just constructed at a cost of $750,000 in New Brunswick, New Jersey, and described as "the finest of its type in the world."

Dedicated to pure science, the institute will be in complete operation this fall. The aim, it was explained, is to create in the medical and biological fields an industry-supported research enterprise comparable to the Bell Telephone and General Electric laboratories in the sphere of physics.

Research activity, already under way, has been organized in four main divisions — experimental medicine, pharmacology, bacteriology and virus diseases, and organic chemistry. In addition, the institute will operate a biochemical laboratory and a medicinal chemistry laboratory. The scientists will continue studies begun in the laboratories with which they were previously associated, and new lines of investigation will be opened up.

Dr. George A. Harrop, formerly associate professor of medicine in Johns Hopkins University and associate physician of Johns Hopkins Hospital, has been appointed director of research in direct charge of the institute. Dr. Harrop will also head the Division of Experimental Medicine.

Dr. Harry B. van Dyke has been chosen head of the Division of Pharmacology. He comes to the institute from Peiping Union Medical College in China, where he was professor and head of the department of pharmacology. He was formerly a professor of pharmacology in the University of Chicago.

Dr. Geoffrey W. Rake, former research associate in the Connaught Laboratories of Toronto University, heads the Division of Bacteriology and Virus Diseases. Dr. Rake has also been on the staff of the Rockefeller Institute.

The head of the Division of Organic Chemistry will be Dr. Erhard Fernholz, formerly of the University of Goettingen and Princeton University, and more recently with the research laboratory of Merck and Company.

Dr. Hans Jensen, formerly of the Institute of Experimental Biology of the University of California, will be associate in charge of the Biochemical Laboratory. Dr. Jensen is a former assistant professor of pharmacology at Johns Hopkins University.

William A. Lott, formerly instructor in chemistry at Rutgers University, will be associate in charge of the Medicinal Chemistry Laboratory.

To provide clinical facilities for the research staff, a plan of hospital affiliation is being worked out by the Division of Experimental Medicine. A free ward of fifteen or twenty beds will be maintained for the observation of patients in connection with various problems being studied at the institute. Under the direction of the Division of Experimental Medicine, new fellowships in medical schools will be established by the institute for the study of cancer, syphilis and hormones. Other fellowships now sponsored by E. R. Squibb and Sons will be continued as part of the institute's program.

The entire research program of the institute, Dr. Harrop pointed out, was determined on the basis of intrinsic scientific interest. "The investigations will not necessarily have an immediate practical purpose in view," he added. "They will be motivated by aims identical with those of investigators in university laboratories and institutes devoted to biological or medical research. Although the primary purpose will be that of pure science, experience in the past has abundantly shown that from just such investigations are most likely to come new discoveries of fundamental significance and practical value.

"The members of the institute staff have been chosen from the ranks of men distinguished in university research who were attracted by the opportunity to carry on their work under conditions similar to those of the university.

"These men of science will labor in a scientific environment unique among research institutions. With its personnel, laboratory building and equipment, the institute is undoubtedly the finest of its kind in the world. The founders confidently look forward to new and durable contributions to the medical and biological sciences and hence to human happiness."

Men of science from all over the country will participate in the dedication of the new laboratory which, it is planned, will take place early in October.

CORRESPONDENCE

U.S.P. ANTI-ANEMIA PREPARATIONS ADVISORY BOARD

To the Editor: I enclose the second announcement of the U.S.P. Anti-Anemia Preparations Advisory Board. This sets forth two very important decisions of the board concerning limitations on concentration and also concerning the addition of other substances to liver extracts. I also include additional names of firms and preparations which have received the approval of the board. This announcement is of great importance to pharmacists, to manufacturers and to physicians.

E. FULLERTON COOK, *Chairman,*
Committee of Revision.

43rd Street and Woodland Avenue,
Philadelphia.

* * *

SECOND ANNOUNCEMENT OF THE *United States Pharmacopoeia* ANTI-ANEMIA PREPARATIONS ADVISORY BOARD

Limit of Concentration for Injectable Liver Extracts. The U.S.P. Anti-Anemia Preparations Advisory Board will not, for the present, assign to any preparations of injectable liver extract a strength greater than 15 units per cubic centimeter because of the possibility of loss of unknown factors of value in the treatment of patients with pernicious anemia during the concentration process.

Action Concerning Mixtures of Other Substances with Liver Extracts. Until evidence has been accumulated to show that substances combined with liver extract do not change its potency or cause deterioration, unitage will not be assigned to mixtures of liver extract with other substances. When a U.S.P. preparation of liver or stomach is mixed with another preparation, the board will not define the preparation as containing liver extract or stomach equivalent to a certain number of U.S.P. units unless the potency of the final mixture as such has been established by appropriate clinical tests.

Caution. It is desirable to establish by preliminary observations on patients with relatively high red-blood-cell counts the approximate potency of any new product before making final observations on patients with lower levels of red blood cells, in order to determine the amount constituting a unit.

Additional Products. The following additional preparations have been assigned unitage by the Anti-Anemia Preparations Advisory Board:

Armour and Company, Chicago, Illinois

Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.25 cc. equals 1 unit (injectable).

American Parentrasol Laboratories, Inc., Bridgeport, Connecticut
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.1 cc. equals 1 unit (injectable).

Julius Blackman Corp., New York, New York
Extractum Hepatis. Dry Liver Extract.
50 capsules (25 gm.) equal 1 unit (oral).

Buffington's, Inc., Worcester, Massachusetts
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
(*a*) 0.1 cc. equals 1 unit (injectable).
(*b*) 0.2 cc. equals 1 unit (injectable).

Difco Laboratories, Inc., Detroit, Michigan
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
1 cc. equals 1 unit (injectable).

Direct Sales Company, Inc., Buffalo, New York
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.2 cc. equals 1 unit (injectable).

Endo Products, Inc., New York, New York
1. Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.1 cc. equals 1 unit (injectable).
2. Extractum Hepatis. Dry Liver Extract.
50 capsules (25 gm.) equal 1 unit (oral).

Henry C. Haist and Company, Kansas City, Missouri
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.1 cc. equals 1 unit (injectable).

Hoy, Salb and Co., Indianapolis, Indiana
1. Liquor Hepatis Purificatus. Parenteral Solution of Liver.
(*a*) 0.1 cc. equals 1 unit (injectable).
(*b*) 0.2 cc. equals 1 unit (injectable).
(*c*) 0.4 cc. equals 1 unit (injectable).
2. Extractum Hepatis. Dry Liver Extract.
50 capsules (25 gm.) equal 1 unit (oral).

C. B. Kendall Co., Inc., Indianapolis, Indiana
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.1 cc. equals 1 unit (injectable).

Kremers-Urban Co., Milwaukee, Wisconsin
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.1 cc. equals 1 unit (injectable).

Lafayette Pharmacal, Inc., Lafayette, Indiana
Extractum Hepatis. Dry Liver Extract.
50 capsules (25 gm.) equal 1 unit (oral).

Eli Lilly and Co., Indianapolis, Indiana
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.5 cc. equals 1 unit (injectable).

A. E. Mallard, Detroit, Michigan
1. Liquor Hepatis Purificatus. Parenteral Solution of Liver.
(*a*) 0.1 cc. equals 1 unit (injectable).
(*b*) 0.2 cc. equals 1 unit (injectable).
2. Extractum Hepatis. Dry Liver Extract.
50 capsules (25 gm.) equal 1 unit (oral).

McNeil Laboratories, Philadelphia, Pennsylvania
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.1 cc. equals 1 unit (injectable).

The National Drug Company, Philadelphia, Pennsylvania
Extractum Hepatis. Dry Liver Extract.
50 capsules (25 gm.) equal 1 unit (oral).

Pro-Medico Laboratories, Inc., Brooklyn, New York
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
(*a*) 0.1 cc. equals 1 unit (injectable).
(*b*) 0.2 cc. equals 1 unit (injectable).
(*c*) 0.4 cc. equals 1 unit (injectable).

Raymer Pharmacal Co., Philadelphia, Pennsylvania
1. Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.2 cc. equals 1 unit (injectable).
2. Extractum Hepatis. Dry Liver Extract.
50 capsules (25 gm.) equal 1 unit (oral).

Sharp and Dohme, Inc., Baltimore, Maryland
Extractum Hepatis. Dry Liver Extract.
9 capsules (4.5 gm.) equal 1 unit (oral).

E. R. Squibb and Sons, New Brunswick, New Jersey
Extractum Hepatis. Dry Liver Extract.
9 capsules (4.5 gm.) equal 1 unit (oral).

The Standard Chemical Company, Inc., Des Moines, Iowa
Extractum Hepatis. Dry Liver Extract.
50 capsules (25 gm.) equal 1 unit (oral).

United States Standard Products Co., Woodworth, Wisconsin
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
0.4 cc. equals 1 unit (injectable).

John Wyeth and Brother, Inc., Philadelphia, Pennsylvania
Liquor Hepatis Purificatus. Parenteral Solution of Liver.
(*a*) 0.1 cc. equals 1 unit (injectable).
(*b*) 0.2 cc. equals 1 unit (injectable).

The board will, as occasion arises, issue supplementary announcements either as a revaluation of the above products based on new clinical data or to make a report on new products which are submitted.

U.S. Pharmacopoeia Anti-Anemia Preparations Advisory Board,
Charles W. Edmunds, M.D., *Chairman,*
William B. Castle, M.D.,
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NOTICES

MASSACHUSETTS MEDICO-LEGAL SOCIETY

In answer to requests from numerous sources, the Massachusetts Medico-Legal Society has arranged for a two-day conference devoted exclusively to medicolegal

topics. The conference will be held at the Mallory Institute of Pathology, Boston City Hospital, on Tuesday and Wednesday, October 4 and 5. The sessions will start at 9 a. m. each day and continue until late afternoon. On Tuesday evening there will be an informal dinner, followed by several short addresses. The topics to be covered at the conference will include talks and demonstrations by medical examiners, judges, lawyers, police officials, chemists and others expert along special lines. It will be open to medical examiners and coroners, and also to physicians and lawyers who may be interested in medicolegal topics. There will be no registration fee but those planning to attend are requested to apply in advance because the accommodations are definitely limited. Application should be sent to Dr. William H. Watters, *Chairman,* Conference Committee, 270 Commonwealth Avenue, Boston.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, September 12

Tuesday, September 13

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

Thursday, September 15

*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.

Friday, September 16

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

Saturday, September 17

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

September 12-14 — American Association for the Study of Goiter. Page 545, issue of March 24, and page 145, issue of July 28.

September 12-15 — American Congress of Physical Therapy. Page 946, issue of June 2.

September 15 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

September 19-22 — Congress of the International Society of Surgery. Page 180, issue of August 4.

September 19-23 — American Association of Railway Surgeons. Page 181, issue of August 4.

September 26 and 27 — Symposium on Occupational Disease. Page 145, issue of July 28.

October 3-15 — Inactive Status Training Course. Page 288, issue of August 25.

October 4 and 5 — Massachusetts Medico-Legal Society. Page 365.

October 7 and 8 — Conference on Rural Medicine. Page 180, issue of August 4.

October 8 and November 15 — American Board of Ophthalmology. Page 282, issue of February 10.

October 17, 1938 and February 20, 1939 — American Board of Internal Medicine, Inc. Page 216, issue of August 11.

October 17-21 — Clinical Congress of the American College of Surgeons, New York City.

October 24-26 — Academy of Physical Medicine, Scientific Session. Washington, D. C.

October 24-November 4 — Graduate Fortnight of the New York Academy of Medicine. Page 181, issue of August 4.

October 25-28 — American Public Health Association. Page 321, issue of September 1.

November 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

March 27-31, 1939 — American College of Physicians. Page 36, issue of July 7.

BOOK REVIEWS

On a New Gland in Man and Several Mammals (Glandulae Parathyreoideae). Ivar Sandström. 44 pp. Baltimore: The Johns Hopkins Press, 1938. $1.00.

Fifty years ago there died in Sweden, Ivar Sandström, the anatomist, who first specifically described the parathyroid glands. The results of his investigations have now become an integral part of the matrix of historical medicine. The interest that the parathyroid glands have aroused of late, especially as to their possible use therapeutically, makes the appearance of this book timely for students of this special subject as well as for practitioners of medicine. The reviewer further believes that this book might well be placed in the hands of every first-year medical student in order that they might gain an insight into the character of investigative work sometimes done by medical students.

An appreciation of the condition of morphological exploration during the last quarter of the nineteenth century may be gleaned from the following words in the opening paragraph of Sandström's paper:

> The existence of a hitherto unknown gland in animals that have so often been a subject of anatomical examination called for a thorough approach to the region around the thyroid gland even in man. Although the probability of finding something hitherto unrecognized seemed so small that it was as much with the purpose of completing the investigation rather than with the hope of finding something new that I began a careful examination of this region. So much the greater was my astonishment therefore when in the first individual examined I found on both sides in the inferior border of the thyroid gland an organ of the size of a small pea, which, judging from its exterior did not appear to be a lymph gland or an accessory thyroid gland, and upon histological examination showed a rather peculiar structure.

Sandström goes on to describe the macroscopic appearance of the glands with regard to position, size, color and variability in form, and their differentiation from lymph nodes and accessory thyroid glands. He suggests the name *glandulae parathyreoideae* for these newly discovered glands because it expresses the characteristic of their being bye-glands to the thyroid gland and for the further reason that embryologically they are analogous to the parovarium and paradidymis. The discussion of the glandular parenchyma and the interstitial tissue is carefully and thoroughly done. The paper has six figures of the gross anatomy and thirteen of the microscopic structure of this organ, using material from the dog, ox, cat, horse, rabbit and man.

A brief account of the life of Sandström and a review of the history of the parathyroid glands appear from the writings of Dr. Johan A. Hammar, professor emeritus of anatomy, University of Upsala. Those fortunate enough to secure a copy of this instructive book will find themselves indebted to Charlotte H. Peters and Dr. John F. Fulton of the Laboratory of Physiology, Yale University School of Medicine, who edited the book and made possible the translation of this paper from the Swedish language by obtaining the services of Dr. Carl M. Seipel.

Quelques Vérités Premières (Ou Soi-Disant Telles) sur les Maladies des Enfants. Robert Debré. 93 pp. Paris: Masson et Cie, 1938. 28 Fr. fr.

Grouped under eleven headings such as examination, hygiene, fevers, and the like, Professor Debré has here given voice to a collection of pediatric aphorisms which are well worth study. The novice especially will find much of value concerning the broad significance of signs and symptoms, hints of practical diagnostic, prognostic or therapeutic import — the products of shrewd observation and sound common sense. The book is meant, of course, to supplement, not to supplant, more formal teachings, but in its secondary field can nevertheless be made to serve admirably.